

# DISEASES of the CHEST

VOLUME XXXVIII

NOVEMBER, 1960

NUMBER 5

## Chronic Pulmonary Tuberculosis in Individuals with Known Previous Primary Tuberculosis\*

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The large scale study of the United States Public Health Service on the prevention of tuberculous meningitis by antimicrobial therapy of children with first infection tuberculosis has focused attention on the age group, infants and young children, in whom early complications are most likely to follow primary tuberculosis. It has long been recognized that the risk of clinical tuberculosis again becomes greater during adolescence and early adult life and that pulmonary tuberculosis is the complication most commonly seen in this older group.

Some clinicians believe that development of chronic pulmonary tuberculosis in adolescence is due largely to a fresh exogenous infection while others are convinced that pulmonary tuberculosis developing after childhood is due to the reactivation of old foci of tuberculous infection. The classic work of Opie<sup>1</sup> and of W. H. Feldman<sup>2</sup> demonstrated that the apparently healed primary parenchymal focus is not a likely source of reinfection. However late reactivation of the initial parenchymal focus has been occasionally described<sup>3,4</sup> and this is particularly apt to happen when cavitation developed within the primary focus. The theory that pulmonary tuberculosis arises chiefly from the intrathoracic nodes derived from the primary infection has long been stressed by Schwartz.<sup>5</sup> Among other possible sources of later pulmonary tuberculosis are the apical seedings described as Simon foci<sup>6</sup> which are created during the stage of primary tuberculosis in which spread by the lymphohematogenous route is predominant.

The pathogenesis of pulmonary tuberculosis in the adolescent and young adult is not just an academic question. On its solution depends the public health approach to its prevention. For if pulmonary tuberculosis occurring in a previously infected adolescent is due largely to reexposure to the disease, then continued supervision of the tuberculous child is unnecessary once an inactive stage is reached. If on the other hand pulmonary tuberculosis appearing during adolescence is largely endogenous in origin then careful supervision including roentgenograms will be necessary to discover the disease in its early and most curable stage.

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TABLE 1—EVIDENCE OF ORIGINAL TB INFECTION IN 113 PATIENTS WITH CHRONIC PULMONARY TBC

Original Diagnosis	First TB Infection	
	Not Seen at Bellevue Hospital	Seen at Bellevue Hospital
Total	19	94
Manifest Primary TBC	12	50
Primary TBC with Calcification	3	41
Positive Tuberculin Reaction Only	4	2
Pleurisy with Effusion	—	1

For many years investigators in the Chest Clinic of the Children's Medical Service of Bellevue Hospital have been interested in this problem and this presentation is intended as a contribution to information about the relationship of primary tuberculosis in the child to chronic pulmonary tuberculosis in the adolescent and young adult. For this purpose the term chronic pulmonary tuberculosis is restricted to fresh evidence of tuberculosis in the lungs of individuals who previously presented evidence of calcified primary tuberculosis or were known to have had first infection tuberculosis followed by an interval with no known manifestation of active pulmonary disease. Locally progressive primary tuberculosis with cavitation is not included nor is bronchogenic spread arising from erosion of a node which was part of the primary complex. In some patients, especially adolescents, it may be difficult on a single examination to distinguish between locally progressive primary tuberculosis and chronic pulmonary tuberculosis. However in a group under follow-up observation it is felt that this distinction is possible.

This report is based on observations of 113 individuals with chronic pulmonary tuberculosis who were observed in the Chest Clinic of the Children's Medical Service of Bellevue Hospital between 1930 and 1959. For all these patients the diagnosis of primary tuberculosis was established while they were in the pediatric age range.

The original diagnoses of the patients are shown in Table 1. Nineteen of the 113 patients were not seen in Bellevue Hospital at the time of the first tuberculous infection. They have been included here because it was possible to obtain earlier records including roentgenograms and thus to diagnose the primary episode retrospectively. It was possible to go back to the manifest primary in 12 patients, to a primary lesion with calcification in 3 patients and to a negative roentgenogram but positive tuberculin reaction in 4 children, one of whom also had extrapulmonary disease.

The remaining 94 patients had first infection tuberculosis when first seen in Bellevue Hospital and developed chronic pulmonary tuberculosis under observation. The roentgenograms of 50 of these patients showed manifest primary disease while those of 42 contained a calcified or calci-

TABLE 2—113 PATIENTS WITH CHRONIC PULMONARY TUBERCULOSIS

	Total	Continental United States		
		White	Non-white	Puerto Rican
Total	113	51	47	15
Male	37	16	14	7
Female	76	35	33	8

lying lesion. Two children with positive tuberculin reactions had no roentgen evidence of pulmonary tuberculosis although one had extrapulmonary disease. One child had pleurisy with effusion and no other roentgen evidence of tuberculosis until he developed chronic pulmonary tuberculosis eight years later. The race and sex of the 113 children is shown in Table 2. The group contains half as many males as females, although during the period of observation the hospital admissions for tuberculosis were about equally divided between the sexes.

Thirty-five of the children who had active primary tuberculosis when they were first seen in the children's chest clinic are of particular interest since in these cases we have the statistical data for a more accurate estimation of the risk of developing chronic pulmonary tuberculosis following a known primary infection during childhood. They were members of a consecutive case group of 1000 children with primary pulmonary tuberculosis assembled from 1930 to 1940.<sup>7</sup> Six hundred twenty-two children had active primary tuberculosis without calcification and of these, 35 developed chronic pulmonary tuberculosis. Charts 1 and 2 are based on this group of 622 active primaries.

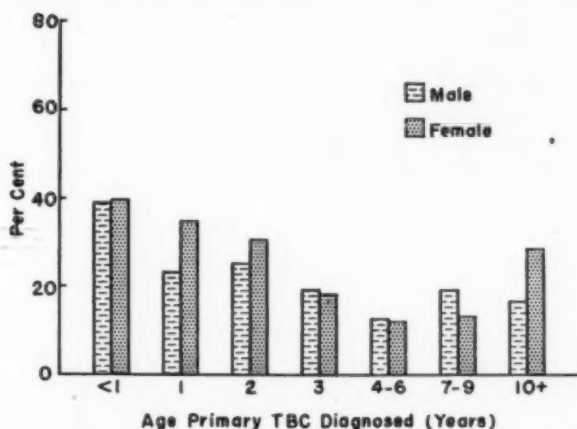


CHART 1: Frequency of fatal complications among 622 children classified by sex and by age at diagnosis of uncalcified primary tuberculosis.

More than 20 per cent of these 622 children died from complications, usually within a year. The proportion of each age-sex group dying from complications is shown in Chart 1. For both sexes the risk of fatal complications such as meningitis and miliary tuberculosis was greatest in infants under a year, decreasing to a low among children who developed primary tuberculosis in the fourth through sixth year of life, and then apparently increasing as adolescence approaches. The number of children in some groups was very small and the differences between the sexes are not statistically significant in any age group.

Chronic pulmonary tuberculosis developed in 35 (7.6 per cent) of the 471 patients who survived primary tuberculosis and were therefore at risk of developing chronic pulmonary tuberculosis. Chart 2 shows the per cent of the survivors in each age-sex group who developed later pul-

monary disease. None of the survivors who had their first infection under one year of age developed chronic pulmonary tuberculosis. When the diagnosis of active primary tuberculosis was made before the age of seven years there is no statistically significant difference in the rate of boys and girls developing later chronic pulmonary tuberculosis. Children who had primary tuberculosis when seven years of age or older not only seem to develop chronic pulmonary tuberculosis at a much higher rate but the rate in girls is higher than in boys. The extremely high rate in girls who had primary tuberculosis when 10 years of age and older may be exaggerated by the relatively few girls with primary tuberculosis seen in this age group.

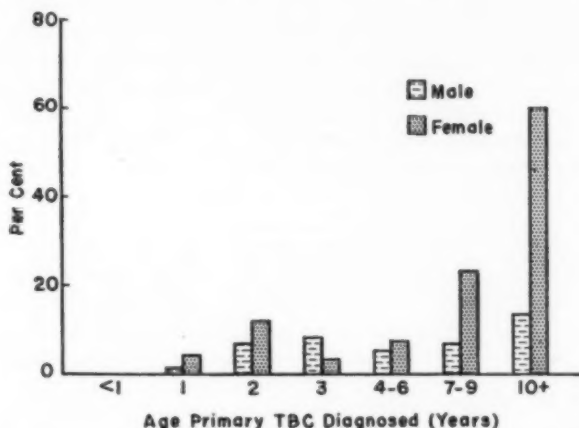


CHART 2: Frequency of chronic pulmonary tuberculosis among 471 survivors of primary tuberculosis classified by sex and by age at diagnosis of uncalcified primary tuberculosis.

The proximity of recently acquired primary tuberculosis to adolescence with all its physiologic changes and stresses may account for the higher rate of chronic pulmonary tuberculosis observed earlier in those, particularly girls, who were over 10 years old when first infected. The age at menarche in relation to the development of chronic pulmonary tuberculosis was known in 65 of the 76 girls. For 20 per cent chronic pulmonary tuberculosis was first diagnosed within a year of menarche, for 40 per cent within two years.

TABLE 3—RELATION OF AGE AT DIAGNOSIS OF PRIMARY TBC TO AGE AT DEVELOPMENT OF CHRONIC PULMONARY TUBERCULOSIS IN 50 PATIENTS

Age Primary Diagnosed	Age Chronic Pulmonary Diagnosed				
	<4	4-6	7-9	10-14	15-19
<4		1	1	8	4
4-6			2	4	1
7-9			2	8	3
10-14				5	5
					3

Correlation of the age at which primary tuberculosis was diagnosed with the age at which chronic pulmonary tuberculosis developed is shown in Table 3. The risk of developing chronic pulmonary tuberculosis is ap-

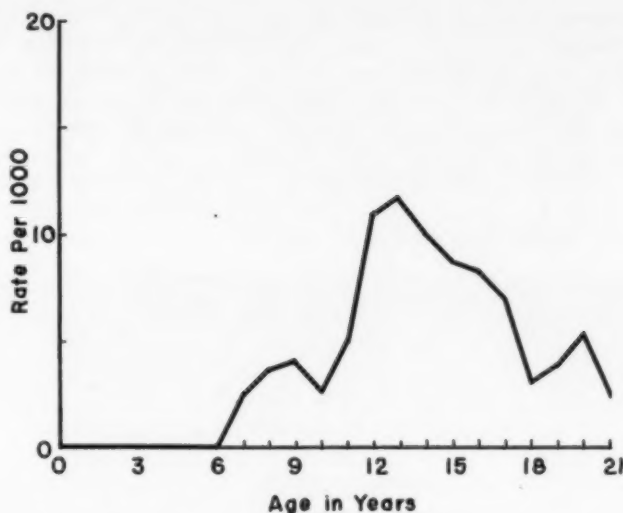


CHART 3: Probability of developing chronic pulmonary tuberculosis in each year of life among survivors of primary tuberculosis.

parently greatest in adolescence irrespective of the age when the primary was diagnosed. The fact that so little chronic pulmonary tuberculosis appears before adolescence suggests that the child's age is a greater factor in the development of chronic pulmonary tuberculosis than the length of time he has had his primary disease.

The annual risk of developing chronic pulmonary tuberculosis among survivors of primary pulmonary tuberculosis in the same group of children is shown graphically in Chart 3. Here again the risk is seen to be greatest during adolescence.

No information could be obtained from this group of patients linking the onset of chronic pulmonary tuberculosis to other situations involving unusual stress such as pregnancy. No boy in this series developed chronic pulmonary tuberculosis while serving in the Armed Forces. Factors such as nutrition undoubtedly play an important part in the pathogenesis of chronic pulmonary tuberculosis. Unfortunately this cannot be measured in this group since the patients came almost entirely from a very low economic level and during many years of the study economic depression or rationing during the war determined their diet.

Seventy-one of the 113 patients gave a definite history of exposure to a case of tuberculosis; this information was usually obtained at the time the primary tuberculosis was diagnosed. An attempt was always made to search for evidence of renewed exposure when the chronic pulmonary tuberculosis was diagnosed but this was found in only seven patients. In four cases individuals who had been the presumed source case for the primary tuberculosis had positive sputum or died of tuberculosis about the time chronic pulmonary tuberculosis in our patient was diagnosed. Three patients were exposed to active tuberculosis in a member of the family other than the source case responsible for the first infection.

The interval between the first diagnosis of primary tuberculosis and that of chronic pulmonary tuberculosis varied from less than one year in five girls (four adolescents and one child of seven years) to more than 20 years, being more than five years in two-third of the patients.

Chronic pulmonary tuberculosis was discovered in two thirds of the patients through a routine roentgenogram, in which tuberculosis was of minimal extent in 75 per cent. Only one-third had symptoms attributable to the pulmonary lesion and some of these may have been elicited after the roentgen evidence was known. The most common symptom was

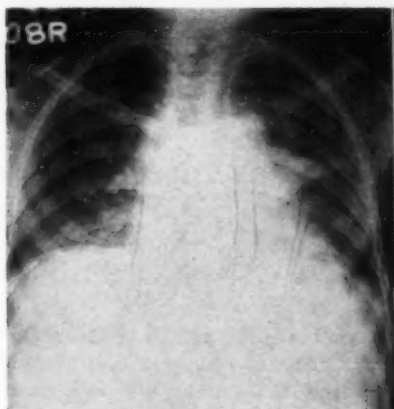


FIGURE 1A



FIGURE 1B



FIGURE 1C

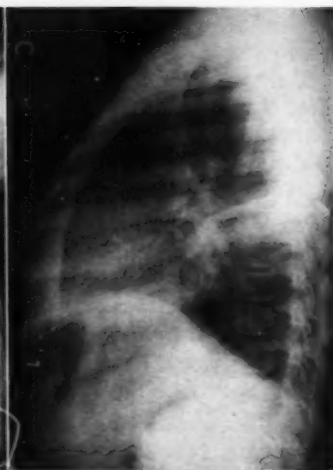


FIGURE 1D

**FIGURE 1:** B.M., aged seven years. A. Roentgenogram of February 27, 1946—enlargement of left hilar area and triangular clouding extending from left heart border outward with apex over third left anterior rib, localized on lateral view (B) in segment number 6. Interpreted as primary tuberculosis left lower lobe. Later roentgenogram showed highlight. Following complete resolution on roentgenogram patient readmitted at age of 10 years. Roentgenogram of December 4, 1949 shows no definite abnormality in parenchyma on PA view (C). Lateral view (D) shows clouding with highlight in segment number 6 of left lower lobe. Tubercle bacilli recovered from gastric lavage.

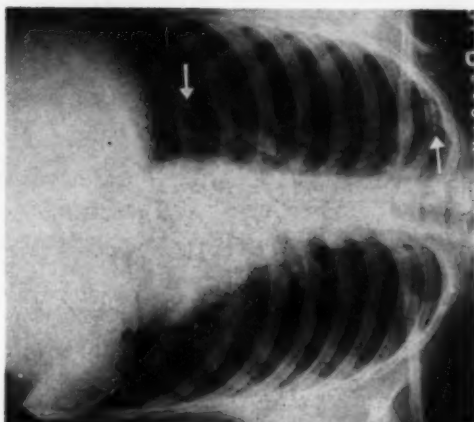


FIGURE 2A

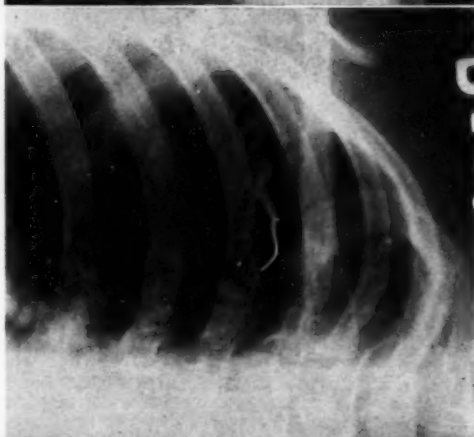


FIGURE 2B

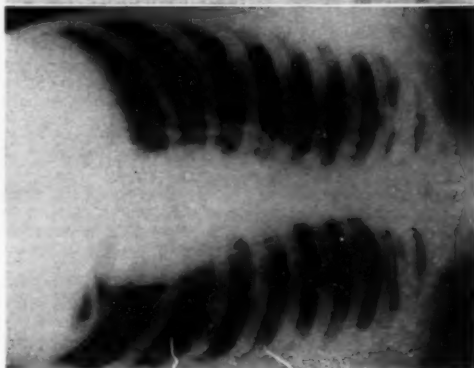


FIGURE 2C

FIGURE 2: A.A., 13 years old. A. Roentgenogram showing calcification of primary pulmonary focus in right apex. B. Enlarged view of apical calcification showing small satellite calcifications. Roentgenogram unchanged for five years. C. Patient 17 years old. Tuberculosis with cavitation adjacent to Simon focus. Tubercle bacilli recovered in culture.

TABLE 4—PRESENT STATUS OF 113 PATIENTS  
WITH CHRONIC PULMONARY TBC

	Male	Female
Total	37	76
Followed to Age 25	20	34
Followed to Age 21-24	6	6
Still under Age 21	2	6
TB Deaths	8	24
Non TB Deaths	1	1
Lost to Follow-up	0	5

cough, occurring in 30 (about 25 per cent). Sixteen patients gave evidence of weight loss and 14 complained of easy fatiguability. Chest pain was present in 11 and fever and hemoptysis each occurred in 10 patients (about 8 per cent). Physical signs were frequently absent. Rales were heard initially in only 25 patients and changes in breath sounds or dullness on percussion were noted in only 15 per cent.

The present status of the 113 patients is shown in Table 4. The death rate from chronic pulmonary tuberculosis in the boys was 21.6 per cent and in the girls 31.6 per cent. Only 20 patients received antimicrobial therapy for chronic pulmonary tuberculosis and in only three instances was treatment given within a year after the diagnosis was established. It seems almost incredible in these days of antimicrobial therapy to note that of the 32 deaths, 22 occurred in patients who were first diagnosed with minimal disease. A few of these received antimicrobial therapy but only in an advanced stage. The five girls lost to follow-up were last seen when 16 to 20 years old and at that time four had inactive disease.

### Discussion

In this group the risk of chronic pulmonary tuberculosis in children with known previous primary infections was greatest during adolescence; the risk was also greatest in children who acquired their primary infection after the age of 7 years. The importance of the sex factor seems obvious. The time relationship to menarche suggests a possible endocrine factor and certainly this subject needs further investigation. Failure to find a fresh source of tuberculosis in all but a few cases also is in favor of exacerbation of old disease rather than superinfection although source cases are often difficult to uncover.

An attempt was made to relate the anatomical site of the primary focus to that of the chronic pulmonary tuberculosis in order to throw some light on the question of the frequency of late exacerbation of first infection disease. In the majority of patients no anatomical relationship between the site of the primary and the chronic pulmonary tuberculosis could be established. Two children with the initial diagnosis of cavitating primary tuberculosis later developed fresh evidence of tuberculosis in the same segment. Roentgenograms of one of these patients are shown in Fig. 1. In addition, chronic pulmonary tuberculosis was first seen around the calcification of the parenchymal component of the primary in nine cases. The probable relation of so-called Simon foci to later evidence of chronic pulmonary tuberculosis was investigated in this group. Seventeen patients showed the characteristic apical calcifications; in five of these patients chronic pulmonary tuberculosis was first observed in the immediate vicinity of the Simon focus. The roentgenograms of one patient are shown in Fig. 2.

### SUMMARY

A group of 113 patients with chronic pulmonary tuberculosis has been presented, all of whom had their primary infection during childhood.

Although admissions to the children's tuberculosis ward were about equally divided between boys and girls, only one-half as many males as females later developed chronic pulmonary tuberculosis.

During follow-up of 622 consecutively seen children with uncalcified primary tuberculosis 35 (7.6 per cent) of the survivors developed chronic pulmonary tuberculosis after intervals of three months to over 20 years.

The risk of chronic pulmonary tuberculosis was greater in children in whom primary tuberculosis developed at the age of seven years or more.

The risk of developing chronic pulmonary tuberculosis was apparently greatest in adolescence irrespective of the age when the primary was diagnosed.

Forty per cent of the cases of chronic pulmonary tuberculosis developed within two years of the menarche.

The majority of instances of chronic pulmonary tuberculosis were found through routine roentgenogram; in 75 per cent the disease was minimal.

Only one-third of the patients had symptoms attributable to the pulmonary lesion at the time of diagnosis of chronic pulmonary tuberculosis.

This series of cases emphasizes the importance of keeping children with primary tuberculosis under long term observation in order to make the earliest possible diagnosis of chronic pulmonary tuberculosis and to gain information about the pattern of its development.

None of the children reported here received specific therapy during the primary phase of the disease. Follow-up of a similar series of children who have been treated by antimicrobial therapy for primary tuberculosis might be instructive.

**ACKNOWLEDGEMENT:** The authors are indebted to Mrs. Shirley H. Ferebee for invaluable suggestions in the preparation of this report.

#### RESUMEN

Se presenta un grupo de 113 enfermos con tuberculosis crónica que tuvieron todos ellos, la infección primaria durante la infancia.

Si bien los ingresos al servicio de niños tuberculosos fué aproximadamente en números iguales entre niños y niñas, el número de niños que mas tarde desarrollaron tuberculosis, fué aproximadamente la mitad del número de niñas que la presentaron.

Durante el seguimiento de 622 niños vistos consecutivamente con tuberculosis primaria no calcificada, 35 (7.6 por ciento) de los sobrevivientes desarrollaron tuberculosis después de intervalos que variaron de tres meses a veinte años.

El riesgo de tener tuberculosis crónica fué mayor en los niños en los que la tuberculosis primaria ocurrió a la edad de siete o mas años.

El peligro de desarrollar tuberculosis pulmonar crónica fué aparentemente el mayor en la adolescencia sin tener en cuenta la edad en que la tuberculosis primaria se diagnosticó.

Cuarenta por ciento de los casos de tuberculosis pulmonar crónica se desarrollaron dentro de los dos años de la menarquia.

La mayoría de los casos de tuberculosis pulmonar crónica se encontraron al hacer radiografía de rutina; en 75 por ciento la enfermedad era mínima.

Sólo un tercio de los enfermos tenían síntomas atribuibles a la lesión pulmonar en el momento del diagnóstico de la tuberculosis pulmonar crónica.

Esta serie hace resaltar la importancia de mantener a los niños con tuberculosis primaria bajo larga observación a fin de hacer lo mas tempranamente posible el diagnóstico de la tuberculosis pulmonar crónica y obtener así información sobre la forma de su desarrollo.

Ninguno de los niños aquí referidos recibieron tratamiento específico durante la fase primaria de la enfermedad. El seguimiento de series similares de niños que hayan sido tratados por la medicación antimicrobiana durante la tuberculosis primaria, sería muy ilustrativo.

#### RESUMÉ

L'auteur rapporte un ensemble de 113 malades atteints de tuberculose pulmonaire chronique, tous ayant fait leur primo-infection pendant l'enfance.

Bien que les admissions au service de tuberculose des enfants fussent à peu près également partagées entre garçons et filles, la moitié seulement aussi bien garçons que filles, furent atteints plus tard de tuberculose pulmonaire chronique.

Au cours d'un contrôle de 622 enfants suivis régulièrement, et atteints de tuberculose primaire non calcifiée, 35 (7.6%) des survivants eurent une tuberculose pulmonaire chronique après un intervalle allant de 3 mois à plus de 20 ans.

Le risque de tuberculose pulmonaire chroniques s'est montré plus grand pour les enfants dont la tuberculose primaire s'était développée à l'âge de 7 ans ou plus.

Le risque de tuberculose pulmonaire chronique fut apparemment plus grand chez les adolescents, sans rapport avec l'âge auquel la tuberculose primaire fut diagnostiquée.

40% des cas de tuberculose pulmonaire chroniques se développèrent pendant les deux ans avant ou après la puberté.

La majorité des cas de tuberculose pulmonaire chronique fut trouvée par l'examen systématique; dans 75% l'atteinte fut minime.

Un tiers des malades seulement eut des symptômes attribuables à la lésion pulmonaire au moment du diagnostic de tuberculose pulmonaire chronique.

Ce groupe de cas met en lumière l'importance de la surveillance à long terme chez les enfants ayant fait leur primo-infection tuberculeuse, pour faire le plus tôt possible le diagnostic de tuberculose pulmonaire chronique et pour obtenir des renseignements sur l'évolution de son développement.

Aucun des enfants dont les cas sont rapportés ici n'avait reçu de traitement spécifique pendant la phase initiale de la maladie. Un contrôle sur un groupe semblable d'enfants qui avaient été traités par le traitement antimicrobien pour tuberculose primaire pourrait être instructif.

#### ZUSAMMENFASSUNG

Es wird berichtet über eine Gruppe von 113 Kranken mit chronischer Lungentuberkulose, die sämtlich ihre primäre Infektion während der Kindheit durchgemacht hatten.

Obwohl die Zugänge auf der Abteilung für Kindertuberkulose ungefähr gleichmässig zwischen beiden Geschlechtern, verteilt waren trat nur bei halbsoviel Männern wie Frauen später eine chronische Lungentuberkulose auf.

Im Verlauf der Nachbeobachtung von 622 nacheinander gesehenen Kindern mit nicht verkalkter Primärtuberkulose kam es bei 35 (7.6%) der Überlebenden zu einer chronischen Lungentuberkulose nach Intervallen von 3 Monaten bis zu über 20 Jahren.

Die Gefahr einer chronischen Lungentuberkulose war größer bei Kindern, bei denen eine Primärtuberkulose im Alter von 7 Jahren oder darüber auftrat.

Die Gefahr Entstehung einer chronischen Lungentuberkulose war augenscheinlich am größten im Jugendalter unbeschadet des Alters, zu dem Primärtuberkulose festgestellt wurde.

40% der Fälle von chronischer Lungentuberkulose entstanden innerhalb von 2 Jahren der Menarche.

Meistens fand man, die chronischen Lungentuberkulosen durch Routine-Röntgenaufnahmen; bei 75% war die Erkrankung minimal. Nur ein Drittel der Kranken hatten Symptome, die auf den Lungenbefund zu beziehen war zum Zeitpunkt der Erkennung der chronischen Lungentuberkulose.

Diese Serie von Fällen betont die Wichtigkeit, Kinder mit Primärtuberkulose für lange Zeit unter Beobachtung zu belassen, um die frühest mögliche Diagnose einer chronischen Lungentuberkulose zu stellen und eine Anschauung davon zu gewinnen, wie sich der Entwicklungsablauf gestaltet.

Keines der Kinder, von denen hier berichtet wurde, war während seiner Phase der Primärerkrankung spezifisch behandelt worden. Es dürften Verlaufsbeobachtungen an einer ähnlichen Reihe von Kindern, die wegen ihrer Primärtuberkulose antimikrobiell behandelt worden waren, sehr lehrreich sein.

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# On the Relief of Bronchospasm and the Induction of Alveolar Hyperventilation:

A Comparative Study of Nebulized Bronchodilators  
by Deep Breathing and Intermittent Positive Pressure\*

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The introduction of aerosol therapy<sup>1,2</sup> with bronchodilator drugs has added much to the management of obstructive pulmonary insufficiency.<sup>3</sup> It is thought to be superior to oral and parenteral administration although comparative controlled data is lacking on this point.<sup>3</sup> Since the combination of aerosol bronchodilators administered with intermittent positive pressure breathing (IPPB),<sup>4</sup> superior results have been obtained to conventional inhalation methods.<sup>5,6,7</sup> Other comparative studies,<sup>8,9</sup> however, have contested the superiority of aerosol-IPPB therapy.

Space does not permit a critical evaluation of each piece of work. There are marked differences in protocol between the methods of administering the aerosols. For instance, sometimes a face mask is used with IPPB and other times a mouth piece. On the other hand with the conventional aerosol bronchodilator studies, a hand nebulizer, a power driven nebulizer, or a mask with a nebulizer has been used. However, in none of these comparative studies<sup>8,9</sup> were arterial bloods drawn. Information relating to arterial blood saturation, pH, and CO<sub>2</sub> tension might shed further light on the comparative effects of simple hyperventilation and IPPB that accompany the administration of a bronchodilator. These measurements seemed to be of great help to the original investigators of IPPB-aerosols.<sup>10,11</sup> It is believed that the increased alveolar ventilation induced by IPPB plays an important role in the mobilization of secretions and the distribution of the aerosol.<sup>4</sup>

## Methods

The patients chosen for this experiment all have symptoms and signs of varying degrees of obstructive pulmonary insufficiency. Their diagnoses include chronic bronchitis, emphysema, and/or asthma. Two thirds have a marked to moderate degree of fixed impairment, as judged by very little change in spirographic values following a bronchodilator. One third have a mild to moderate degree of impairment which may fluctuate from day to day and show large increases in spirographic values following a bronchodilator.

There are 33 males and 3 females. Their ages range from 16 to 76 with a mean of 56. The average height is 68 inches and average weight 145 lbs.

\*Presented at the 25th Annual Meeting, American College of Chest Physicians, Atlantic City, June 3-7, 1959.

\*\*Scripps Clinic and Research Foundation.

The design of this experiment is to compare the efficacy of two methods of administering an aerosol bronchodilator.\* The first method is by deep breathing and intermittent nebulization (DB). The second method is by intermittent positive pressure breathing (IPPB). The relative degree of alveolar hyperventilation induced during the two procedures is judged by following arterial blood saturation, pH, and CO<sub>2</sub> tension.

The protocol calls for the deep breathing and intermittent nebulization study one day followed in one to five days by the IPPB. Separate control spirographic analyses and blood studies are done prior to each study. Control spirographic measurements\*\* in the standing position of vital capacity (V. C.), absolute three second timed vital capacity (3 sec. T.V.C.), and maximum breathing capacity (M.B.C.) are made. The control arterial blood samples are then taken after a period of 15 to 20 minutes rest. After ten minutes of bronchodilator drug administration, the arterial blood sample is repeated and the indwelling arterial needle withdrawn. The post-treatment spirogram is then repeated within twenty minutes after the bronchodilator drug is discontinued.

In 36 patients, spirographic determinations are made according to the above protocol. In only 15 are arterial bloods drawn.

The physical appearance and mechanics of the deep breathing - intermittent nebulization setup are shown in Figure 1. It should be noted that a mouth piece and nose clip are used as in the IPPB experiments to insure introduction of maximal concentration of the medication into the pharynx and presumably into the tracheobronchial tree. A rhythmic timing by an accompanying metronome is used to insure one second for maximal inspiration, and three seconds for maximal expiration. During

\*Isoproterenol — "Isuprel"

\*\*13.5 L Collins Spirometer with Low Resistance Valves.<sup>11</sup>

#### DEEP BREATHING and INTERMITTENT NEBULIZATION

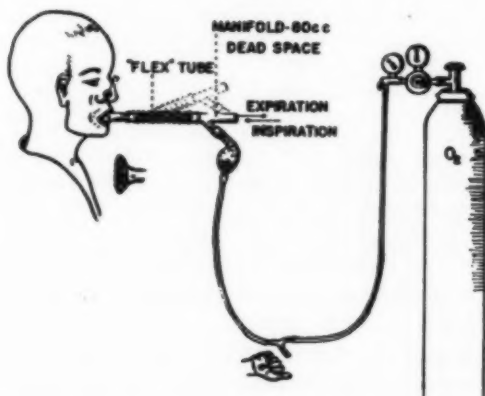


FIGURE 1: The thumb is placed over the glass y tube during inspiration (or kept there during inspiration — expiration depending on patient cooperation). The flex tube allows the manifold and nebulizer to be raised to prevent reflux of saliva. For rest of details, see text.

TABLE 1 — SPIROGRAPHIC DATA

Case No.	Height inches	Weight lbs.	Age yrs.		Vital Capacity cc.		3 sec. T.V.C. cc.		M.B.C. Control	L/min. post R <sub>x</sub>
					Control	post R <sub>x</sub>	Control	post R <sub>x</sub>		
909	72	128	33	DB	2164	4371	1351	2884	22	77
			M	IPPB	2343	3875	1396	2433	20	38
911	70	165	53	DB	3724	3768	3095	3499	109	139
			M	IPPB	3679	3588	3140	3499	92	125
907	67	123	56	DB	4037	4037	2511	2691	62	80
			M	IPPB	4055	4055	2614	2794	62	75
922	69	175	56	DB	4191	4191	3560	3650	110	117
			M	IPPB	4281	4146	3244	3425	110	125
928	65	106	45	DB	1963	2052	1249	1695	27	44
			F	IPPB	2063	2154	1256	1704	27	50
978	65	218	29	DB	2945	3212	1785	1963	22	52
			F	IPPB	2498	2945	1695	2320	25	57
992	71	203	50	DB	4104	4817	2766	3302	62	81
			M	IPPB	3613	4753	2409	3123	57	85
943	70	203	65	DB	3926	4194	3480	3659	81	94
			M	IPPB	3748	4014	3302	3302	94	101
977	68	172	46	DB	4306	4486	1525	2243	25	32
			M	IPPB	3679	4396	1345	2332	17	37
645	72	172	64	DB	5114	5203	4576	4754	156	154
			M	IPPB	3996	4885	3286	3952	78	123
960	72	192	70	DB	4194	4194	2498	2899	49	62
			M	IPPB	4082	4261	2736	2781	57	62
998	72	136	56	DB	3875	3965	1938	1938	35	40
			M	IPPB	3524	3524	1918	1963	34	32
967	61	68	16	DB	2677	2677	2231	2588	49	64
			M	IPPB	1525	2511	1032	2108	15	50
1042	70	154	43	DB	4041	4530	3197	3907	86	123
			M	IPPB	3970	4327	2632	4014	66	118
1027	69	165	56	DB	3140	4174	3064	3152	76	78
			M	IPPB	3140	3679	2422	2870	55	79
1056	68	147	62	DB	2498	3212	1338	1695	27	34
			M	IPPB	2721	3167	1561	1874	32	42
1069	65	118	64	DB	2989	2855	1918	1874	37	37
			M	IPPB	3006	3095	1929	2018	37	40
1060	67	116	70	DB	2945	3034	1382	1606	22	27
			M	IPPB	2766	3167	1428	1874	22	32
1055	70	164	76	DB	3229	3454	2243	2243	37	47
			M	IPPB	3480	3480	2231	2320	47	52
1112	69	160	64	DB	3331	3596	1909	2309	39	56
			M	IPPB	2886	3730	1909	2265	42	49
1135	65	127	70	DB	2238	2511	991	1256	15	15
			M	IPPB	2243	2511	1122	1256	15	15
1155	67	153	60	DB	1929	2826	1032	1481	22	32
			M	IPPB	3335	3515	1487	2478	32	47
1156	66	125	56	DB	4037	3902	2332	2826	59	69
			M	IPPB	3544	3947	2198	2736	42	62
1171	69	168	50	DB	2154	3320	1077	2063	21	32
			M	IPPB	2915	3229	1929	2154	42	42
1180	69	102	60	DB	2736	3140	1749	1884	36	41
			M	IPPB	2736	2669	1749	1951	31	37
2028	64	87	65	DB	1785	1829	936	936	15	17
			M	IPPB	1731	2132	932	1067	20	29
2039	69	182	47	DB	4371	4885	3346	4485	49	74
			M	IPPB	3881	4194	2431	2899	30	33
1094	71	145	50	DB	4174	4553	2220	2298	31	39
			M	IPPB	4041	4307	2132	2398	32	37
2082	62	77	64	DB	1171	1061	796	884	12	12
			F	IPPB	1132	1067	883	888	14	14
1216	69	106	53	DB	1821	2709	976	1160	11	12
			M	IPPB	1963	2320	1026	1071	11	12
1334	67	134	57	DB	4082	4506	3140	3346	84	94
			M	IPPB	4037	4486	3167	3365	71	97
1211	69	144	70	DB	3786	3786	2072	2208	45	50
			M	IPPB	3736	4281	2028	2501	47	54
1493	70	117	63	DB	3014	4149	2553	2588	51	64
			M	IPPB	3926	4283	2298	2632	54	67
1488	66	160	72	DB	2704	3199	1757	2253	42	64
			M	IPPB	2736	3185	1637	2154	45	52
1399	73	142	55	DB	3741	5472	2268	3588	32	60
			M	IPPB	4304	4639	2592	3034	45	55
1352	71	164	55	DB	1777	2797	1022	1510	15	25
			M	IPPB	2154	2975	1598	1731	17	26

inspiration, the nebulizer is started. Expiration is limited to 3 seconds to avoid fatigue. During IPPB, the breathing pattern established voluntarily by the patient is used. He is cautioned to breathe slowly and blow out his expiratory reserve before inhalation.

### Results

The original data is presented in Tables 1 and 2 from which Figures 2 through 4 are produced.

The control spirographic data in terms of normal predicted values<sup>11</sup> are shown in Figure 2. Although control spirographic values on any one subject are observed to vary (see Table 1), as a group our 36 patients had comparable control data before DB and IPPB maneuvers. Those subjects whose control values fluctuated more than 10% were not eliminated as they were in Kory's study.<sup>3</sup>

### Spirographic Data

In Figure 3, the per cent change of absolute values of V.C., 3 sec. T.V.C., and M.B.C. after treatment are shown. The increase is the same for both methods of administration of the bronchodilator. The IPPB values compare favorably with those reported previously.<sup>3,10</sup>

However, in the comparative studies of Wu et al.,<sup>7</sup> the percentage increase 20 minutes after a power driven aerosol by mask is significantly lower than that obtained with IPPB. This difference becomes even more pronounced one hour later. Our study is more similar in protocol to theirs than any of the other comparative studies.<sup>8,9</sup> The one difference that might account for dissimilar results is the method of administering the power driven aerosol.

In their study, a simple face mask with aerosol spray entering a side arm is used. The patient inhaled such medication with continuous tidal ventilation. This is in contrast with this experiment.

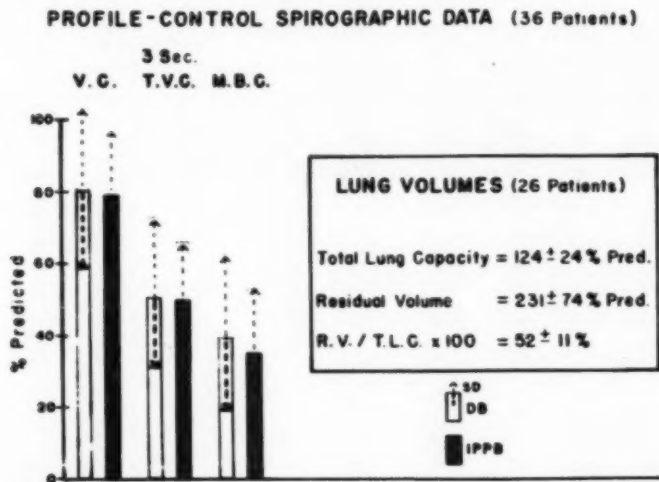


FIGURE 2: The standard deviations about the means are shown as the dotted lines with arrows above and below the top of each bar. The Residual Volume is done according to a modification of the closed circuit helium dilution method (13).

TABLE 2 — ARTERIAL BLOOD DATA

Case No.		pH		Calc. pCO <sub>2</sub> mm Hg		CO <sub>2</sub> Content mM/L		Saturation %	
		Control	post R <sub>x</sub>	Control	post R <sub>x</sub>	Control	post R <sub>x</sub>	Control	post R <sub>x</sub>
1069	DB	7.37	7.33	48	53	24.7	24.2	90.8	96.7
	IPPB	7.35	7.38	52	43	25.5	22.0	90.7	100.0
1112	DB	7.33	7.37	49	38	22.2	18.2	89.6	100.0
	IPPB	7.45	7.59	36	22	20.8	16.1	94.6	100.0
1135	DB	7.32	7.37	53	44	24.0	21.5	84.0	92.7
	IPPB	7.37	7.40	47	43	23.3	22.7	83.6	94.0
1155	DB	7.36	7.38	57	47	28.0	23.6	87.6	96.7
	IPPB	7.37	7.45	48	35	24.5	20.5	90.1	99.3
1171	DB	7.37	7.40	51	39	23.6	19.2	87.5	99.0
	IPPB	7.34	7.40	54	41	24.4	19.5	86.5	100.0
2028	DB	7.41	7.39	49	48	25.6	24.3		
	IPPB	7.43	7.44	50	43	26.7	23.8		
2039	DB	7.40	7.56	40	24	20.7	17.2		
	IPPB	7.40	7.44	39	32	20.8	18.4		
2082	DB	7.42	7.45	53	35	29.8	27.2	92.3	99.5
	IPPB	7.42	7.48	48	34	26.7	21.2	89.0	90.6
1216	DB	7.36	7.36	46	50	22.1	23.1	86.9	90.5
	IPPB	7.30	7.42	53	34	21.9	20.0	72.4	96.5
1334	DB	7.40	7.37	43	45	23.2	22.8		
	IPPB	7.45	7.45	38	35	22.8	21.9		
1211	DB	7.50	7.56	40	31	25.8	23.0	90.2	94.2
	IPPB	7.53	7.65	36	22	26.1	21.9	89.8	94.8
1493	DB	7.40	7.52	41	25	22.6	17.6	90.5	94.0
	IPPB	7.41	7.53	40	24	22.2	17.4	90.1	95.2
1488	DB	7.39	7.44	41	34	22.2	19.7	92.5	91.4
	IPPB	7.41	7.47	40	30	22.8	18.5	90.0	94.5
1399	DB	7.42	7.44	38	33	21.0	19.2	95.1	98.0
	IPPB	7.39	7.46	43	32	22.5	19.2	92.2	97.2
1352	DB	7.42	7.44	50	47	27.4	25.2	81.6	86.5
	IPPB	7.42	7.47	52	42	26.4	23.7	84.6	100.0

## Arterial Blood Data

In Figure 4, arterial blood changes before and after the bronchodilator are shown. It should be recalled that pure oxygen is used with the IPPB-aerosol and that pure oxygen drives the aerosol in the DB experiment.

% CHANGE (ABSOLUTE FIGURES)  $\dot{V}$  TREATMENT

Deep Breathing vs IPPB  
36 Patients

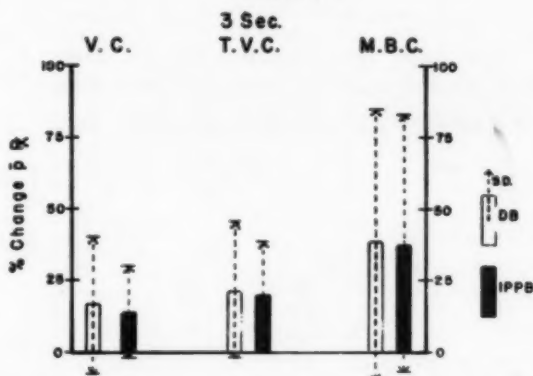


FIGURE 3: The standard deviation about the mean is shown as in Figure 1. For details, see text.

In this latter experiment, the pure oxygen stream is mixed with room air and in all probability, results in an inspiratory mixture of some 50 per cent oxygen or greater.

The results are somewhat surprising. The greater rise in pH with IPPB is the only significant difference in the four arterial blood factors which indicate superior alveolar hyperventilation. Perhaps with more data and a different experimental approach, the superiority of IPPB over DB might have been more clearly defined.

It may be that the significant difference in pH change between DB and IPPB does not represent alveolar ventilatory differences. The mechanical effort for respiratory cripples and the physiologic work required for 10-15 minutes of the deep breathing maneuver is great. Such effort may drive the pH in opposite direction from respiratory alkalosis to metabolic acidosis. In other words, the difference of .03 pH units (+0.07 IPPB and +0.04 DB) may represent metabolic rather than ventilatory differences.

### Discussion

In analysing the differences between our results and those published by Wu,<sup>7</sup> we have already commented on the method of administering a bronchodilator. It also occurred to us that there might be a difference between patients with a relatively "reversible" degree of pulmonary insufficiency and those with an irreversible or fixed insufficiency. The spiographic data were rearranged accordingly.

The 24 patients with a "fixed" degree of pulmonary insufficiency showed no difference in the degree of improvement following a bronchodilator regardless of whether it was administered by DB or IPPB. (V. C. + 9 per cent DB vs. + 9 per cent IPPB; 3 sec. T. V. C. + 11 per cent DB vs. + 13 per cent IPPB; M. B. C. + 19 per cent DB vs. + 21 per cent IPPB.)

The 12 patients (case #909, 928, 967, 977, 978, 992, 1042, 1155, 1171, 1216, 1399, 1352. Table 1) with an easily reversible degree of pulmonary insufficiency also showed no statistical difference in the degree of improvement following a bronchodilator regardless of whether it was administered by DB or IPPB. (V. C. + 31 per cent DB vs. + 22 per cent IPPB; 3 sec. T. V. C. + 32 per cent DB vs. + 24 per cent IPPB; M. B. C. + 66 per cent DB vs. + 62 per cent IPPB.)

The deep breathing method of administering a bronchodilator has proven most helpful in mild to moderate degrees of obstructive pulmonary insufficiency, as defined in the introduction above. However, the effort required for this maneuver is too great for the severe respiratory cripple. In such instances and when respiratory acidosis and coma are present, IPPB remains the treatment of choice for administering a bronchodilator and inducing alveolar hyperventilation.

### ARTERIAL BLOOD CHANGES $\bar{p}$ BRONCHODILATOR

Deep Breathing (DB) vs Positive Pressure (IPPB)

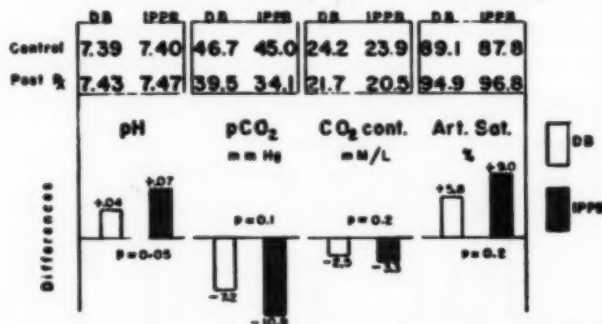


FIGURE 4: The probability values ( $p$ ) for comparing the absolute changes of DB vs. IPPB are designated at the base of each pair of bars. They are from left to right, for pH 0.05, for  $PCO_2$  0.1 for  $CO_2$  content 0.2, for art. sat. 0.2.

## SUMMARY

1. Under the acute conditions of this experiment, the relief of bronchospasm is the same with aerosols of isoproterenol whether administered by deep breathing or with intermittent positive pressure breathing.

2. The degree of alveolar hyperventilation induced by the two methods seemed to be comparable except for the greater rise in arterial pH with IPPB.

**ACKNOWLEDGMENT:** The author wishes to express his thanks to Betty Schitel, laboratory technician, currently in Milwaukee, Wisconsin, for her help in the initial phase of the work, and to Kachina Conley, typist and secretary.

## RESUMEN

1. Bajo las condiciones agudas en que se hizo este experimento, el alivio del espasmo bronquial es igualmente obtenido con aerosoles de isoproterenol ya se administren con inspiración profunda o con respiración bajo régimen de presión positiva intermitente.

2. El grado de ventilación alveolar producido por los dos métodos pareció comparable salvo que hubo una elevación mayor del pH con la respiración bajo presión positiva intermitente.

## RESUMÉ

1. Dans les conditions précises de l'expérimentation de l'auteur, le soulagement du bronchospasme est le même avec des aérosols d'isoproterenol administrés soit en respiration profonde soit sous respiration en pression positive intermittente.

2. Le degré d'hyperventilation alvéolaire produit par les deux méthodes semble comparable, sauf pour l'élévation plus grande du pH artériel sous respiration en pression positive intermittente.

## ZUSAMMENFASSUNG

1. Unter den verschärften Bedingungen dieses Experimentes ist die Behebung eines Bronchospasmus mit Aerosolen von Isoproterenol die gleiche, unbeschadet dessen, ob die Verabfolgung nur durch tiefe Atemzüge geschah oder durch intermittierende positive Druckatmung.

2. Das Ausmaß der durch die beiden Methoden bewirkten alveolären Hyperventilation scheint vergleichbar, abgesehen von dem größeren Anstieg des arteriellen pH bei intermittierender positiver Druckatmung.

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## Resection of Descending Thoracic Aorta and Replacement while Maintaining a Continuous Blood Flow

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Resection of the descending thoracic aorta for aneurysm or other lesions presents a number of technical problems and, in addition, a number of questions with respect to long-term results in such procedures.

It is a known fact that interruption of the circulation to important organs such as the spinal cord and kidneys for relatively short periods of time may result in paresis of the lower extremities and anuria. This means that the procedure should either not exceed the time limit imposed or else circulation to the vital areas must be maintained in some other way, such as by shunting techniques with or without the use of a pump.

The technique presented in this report is one in which the anastomosis can be performed while maintaining a continuous blood flow without the use of a shunt and also avoiding the interruption of blood supply to important organs except for a very brief period of time averaging two to three minutes while the prosthesis is applied.

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This work was supported in part by the E. F. Andrews Fund for Thoracic Surgery.

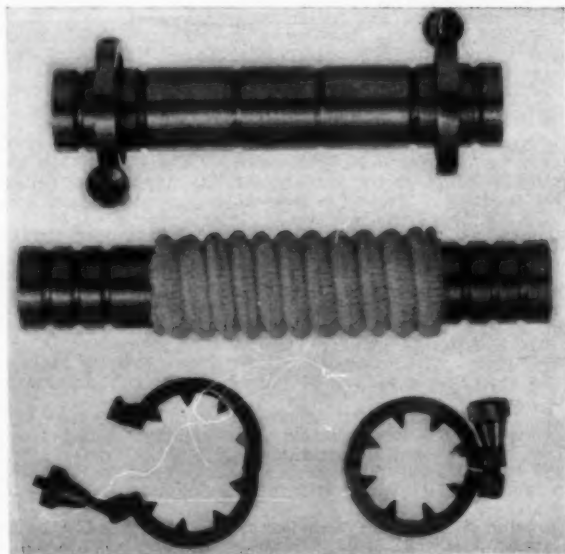


FIGURE 1: Metallic prosthesis. Top — shows the grooves and the clips applied and secured in place. Middle — the Teflon graft mounted around the middle portion of the tube. Lower — clips.

### Method and Procedure

The method employed in this procedure entails the use of a special prosthesis which consists of: 1. a metallic stainless steel tube with furrows at each end and 2. two clamps based on the principle of multiple point pressure so that, while it will apply the necessary pressure needed to prevent leakage of blood around the tube, it will not traumatize the vessel wall (Fig. 1).

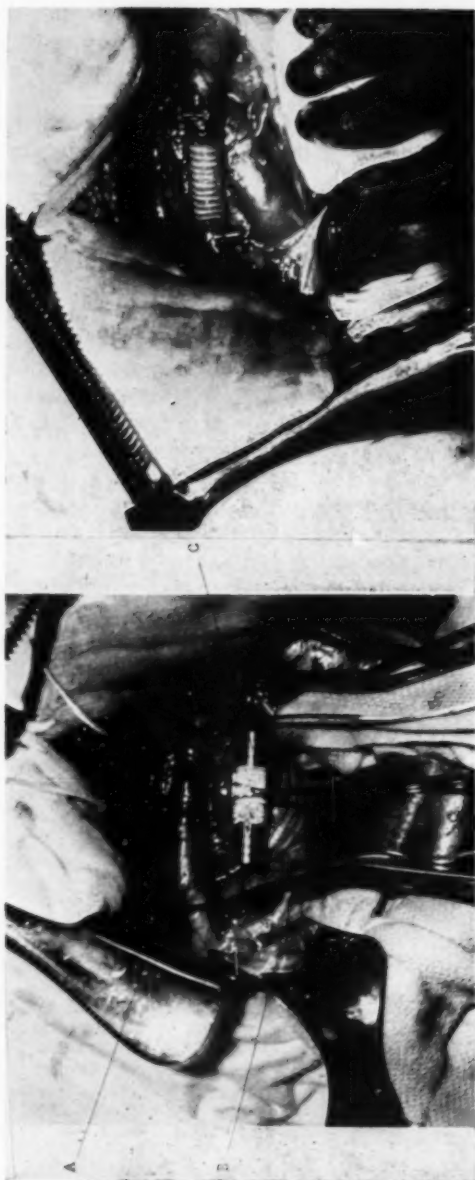
The clamp will only close completely if the teeth are lodged in the furrow. It is then secured in place by a hinged handle and screw.

The left thoracic cavity was entered through the sixth or seventh intercostal space. The lung was retracted medially and the descending thoracic aorta exposed. The intercostal vessels were dissected free (over the upper seven intercostal spaces) and were doubly ligated and divided. A straight Potts clamp was applied proximally and another distally with complete occlusion of the blood flow and the segment of aorta was excised. The metallic tube, around which the Teflon graft had been mounted in such a way that the graft would only occupy the middle portion of the tube (Fig. 2a) was then applied. The end of the tube was passed through the cut end of the vessel. The clamp was applied around the aorta which would fasten the vessel wall around the tube and this was secured in place by means of the screw. The same procedure was carried out distally. The distal and the proximal Potts clamps were then disoccluded, thus allowing the blood to flow through. The Teflon graft was anastomosed to the vessel using continuous silk sutures. The graft around the tube was grasped with a Babcock clamp, avoiding migration of the tube after the clamps were released.

To remove the tube from inside the vessel, two Potts clamps were applied distally and proximally and a longitudinal incision about 1 cm. was made on the anterior wall of the vessel. The tube was then grasped with

TABLE 1 — RESULTS FOLLOWING RESECTION OF THORACIC AORTA AND REPLACEMENT WHILE MAINTAINING ITS CIRCULATION IN 18 DOGS

Tag No.	Date of Operation	Postoperative Course Died      Sacrificed	Results and Findings
372	8/ 5/59	12 days	Thrombosis of distal abdominal aorta, bifurcation and branches; graft patent
396	8/ 6/59	58 days	No complication; graft patent
423	8/ 7/59	Immediately postoperative	Anesthesia shock; graft in good position
931	8/12/59	43 days	No complication; graft patent
977	8/13/59	183 days	No complication; graft patent
595	8/15/59	177 days	No complication; graft patent
547	8/19/59	182 days	No complication; graft patent
517	8/21/59	191 days	No complication; graft patent
540	8/24/59	164 days	No complication; graft patent
403	8/27/59	52 days	No complication; graft patent
834	10/27/59	67 days	No complication; graft patent
892	10/28/59	36 days	No complication; graft healed; moderate reaction
820	10/29/59	15 days	No complication; graft patent
518	11/ 4/59	63 days	No complication; graft patent
838	11/ 9/59	56 days	No complication; graft patent
856	11/11/59	52 days	No complication; graft patent
878	11/16/59	14 days	No complication; graft patent
866	11/17/59	43 days	No complication; graft patent

**FIGURE 2a****FIGURE 2b**

**FIGURE 2a:** Shows aorta (A) dissected free; a proximal and a distal Potts clamp (B) applied; the metallic prosthesis (C) with the graft mounted. **FIGURE 2b:** The Teflon graft sutured to aorta. The metallic tube and clips still in place. Blood flow maintained throughout the procedure.

a Kocher clamp and removed. A curved Potts clamp was applied at the incision site and the two Potts clamps removed. This allowed blood to flow normally while the incision in the vessel was sutured with continuous silk sutures; then the curved Potts clamp was removed (Fig. 2b).

### *Results*

Experiments were made on 18 dogs (Table 1). In the first dog there was migration of the tube distally due to a technical error and this dog died on the 12th postoperative day. Post mortem examination showed thrombosis in the distal abdominal aorta and its bifurcation and branches distally. Another dog died at the end of the procedure due to anesthesia shock. Post mortem examination revealed the graft and sutures to be in good position. The remaining 16 dogs did very well postoperatively and are living from two to five months following surgery. They have behaved normally and have presented no signs nor symptoms of neurological or vascular complications. The EKG and EEG were normal during and after completion of the procedure. Aortogram showed the graft to be in excellent position without narrowing or distortion at the anastomosis site and with perfect flow distally (Fig. 3).

### *Discussion*

The results of resection and Teflon graft replacement of the thoracic aorta in dogs by using a special prosthesis and clamps are believed to compare favorably with any of the experimental reports which have come to our attention.



FIGURE 3: Aortogram five months' postoperative demonstrating excellent position and flow at the anastomosis site.

Anastomosis can be accomplished while maintaining a continuous blood flow during the whole procedure, except for a very brief period, averaging between two to three minutes, without resorting to a bypass procedure or hypothermia.

The operating time is greatly shortened by this procedure and end-to-end anastomosis is definitely superior to a bypass. The technique could be applied anywhere in the vascular tree by using the appropriate size of prosthesis and clamps. It would seem reasonable that the method presented here will find clinical application.

#### SUMMARY

A method is described for resection and a Teflon heterograft replacement of the thoracic aorta in dogs by using a special prosthesis and vascular clamps in conjunction with brief interruption of the circulation. The method was used in 18 dogs. One dog died on the 12th postoperative day as a result of a technical error and another died at the end of the procedure due to anesthesia shock. The remaining dogs survived and remained in good health with no evidence of physiologic impairment of nervous or cardiovascular function from five weeks to six months following operation. X-ray film of the chest and aortogram have demonstrated excellent position and function.

#### RESUMEN

Se describe un método para la resección de la aorta torácica, substituyéndola con un injerto heterogéneo de Teflon, en los perros, por medio de una prótesis especial y clamps vasculares combinados con una interrupción breve de la circulación. Se usó el procedimiento en 18 perros. Uno de ellos murió a los 12 días de la operación a consecuencias de un error técnico y otro murió al terminar el procedimiento, debido a choque anestésico. Los demás perros sobrevivieron en buena salud sin muestras de daño fisiológico o trastorno nervioso o cardiovascular, de cinco semanas a seis meses después de la operación.

Las radiografías del tórax y los aortogramas, han demostrado la excelente posición y la función.

#### RESUMÉ

Les auteurs décrivent une méthode de résection de l'aorte thoracique chez les chiens, et son remplacement par hétérograffie de teflon, en utilisant une prothèse spéciale et en pratiquant un clampage associé à une brève interruption de la circulation. La méthode fut utilisée chez 18 chiens. Un chien mourut le douzième jour après l'opération à la suite d'une erreur de technique, et un autre mourut à la fin de l'intervention par choc anesthésique. Les autres chiens survécurent et restèrent en bonne santé sans preuve de trouble physiologique de la fonction nerveuse ou cardiovasculaire cinq semaines à six mois après l'opération. Des films radiologiques du thorax et des aortographies ont fait la preuve que le résultat était excellent au point de vue général et fonctionnel.

#### ZUSAMMENFASSUNG

Beschreibung einer Methode zur Resektion der thorakalen Aorta bei Hunden mit Ersatz durch ein Fremd-Transplantat mit Teflon unter Verwendung einer Spezial-Prothese und Gufäßklemmen in Verbindung mit einer kurzen Unterbrechung des Kreislaufes. Das Verfahren wurde bei 18 Hunden zur Anwendung gebracht. Ein Hund starb am 12. Tag nach der Operation infolge eines technischen Irrtums, und ein anderer starb am Ende des Eingriffes durch einen Anästhesie-Schock. Die restlichen Hunde überlebten und blieben in guter Verfassung ohne Anzeichen einer physiologischen Schädigung ihrer nervlichen oder kardiovaskulären Funktion während einer Zeit von 5 Wochen bis 6 Monaten nach der Operation. Thoraxröntgenaufnahmen und Aortogramme zeigten hervorragende Lage und Funktion.

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## Hiatal Hernias in Children: Special Reference to the Short Esophagus\*

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Esophageal hiatal hernias are relatively uncommon in children. Since 1950 we have seen 20 children with hiatal hernias at the Mayo Clinic. During the same period the diagnosis of esophageal hiatal hernia has been made in more than 17,000 adults, and the overwhelming majority of these adults were in the older age group. In this communication we shall review the cases and the roentgenographic findings of some of these 20 children in order to illustrate the types of hernias encountered and the problems that they present. Also, we shall discuss our experience in the management of hiatal hernia and its complications in infants and children.

At the time of their first registration, all 20 children were 14 years of age or less. Although most of them were seen early in life, some of them have been followed for as long as 20 years. In all patients, at least part of the stomach was herniated through the esophageal hiatus of the diaphragm into the thorax.

By means of the classification of Akerlund,<sup>1</sup> the hernias may be classified as paraesophageal hernias, sliding hernias, or hernias of the short esophagus type. In the paraesophageal or rolling hernia, the esophagus is of normal length and extends to the esophageal hiatus but part or all of the stomach is herniated into the thorax through the hiatus. The sliding or gastroesophageal hernia likewise has a portion of stomach within the thorax. However, the esophagogastric junction is located above the esophageal hiatus and the lower portion of the esophagus is usually tortuous and redundant. The term "short esophagus" should be reserved for those patients who have an actual anatomic shortening of the esophagus. In these patients part of the stomach must reside in the thorax in order to provide for esophagogastric continuity.

### *Clinical Material*

In classifying the esophageal hiatal hernias of the 20 children, we found that two had hernias of the paraesophageal type, one had a hernia of the sliding type and the remaining 17 apparently had a short esophagus with intrathoracic stomach. Although we are primarily concerned with the 17 patients having short esophagus, we should like to review briefly the cases of the other three patients.

**Paraesophageal Hernia.** — Both patients having this type of hernia were male infants, and both were admitted as emergency patients with signs of obstruction of the gastrointestinal tract. They required immediate surgical intervention, and in both patients the entire stomach was

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herniated through the esophageal hiatus into the thorax. In neither patient was there any abnormality of the esophagus itself. One of the children was less than a week old and suffered postoperative complications including bilateral pneumothorax and diarrhea. Despite these unfavorable complications the child appeared to be making a satisfactory recovery. Quite unexpectedly, he died about a week after surgical intervention and, unfortunately, necropsy was not permitted.

The other child was slightly more than 4 months of age when obstructive symptoms developed. He had been vomiting for 24 hours when he was admitted to the hospital. It was thought that he might have a malrotation of the intestine, but operation showed that his entire stomach was displaced into the left side of the thorax. Although the child's postoperative course was stormy, he made a good recovery and returned some 14 months later for repair of an incisional hernia. Recent correspondence with this child's physician reveals that he is well and apparently normal at 9 years of age.

*Hiatal Hernia of Sliding Type.* — A 3-month-old boy was registered at the clinic on May 14, 1956. His mother stated that he would regurgitate his feedings unless she held him in an upright position for 2 to 3 hours after he was fed. Roentgenograms of the stomach and esophagus at the original visit did not show any abnormality and it was felt that the child had the clinical picture of chalasia. After some discussion it was decided to carry out a conservative program. The mother was instructed to keep the child in an erect position after eating and to elevate the head of his bed. He continued to have trouble, however, and returned to us on December 4, 1956. At this time a hiatal hernia was demonstrated by roentgenography. Esophagoscopy disclosed severe ulcerative esophagitis. Surgical treatment was recommended, and although we were concerned about the length of the esophagus, a satisfactory repair was obtained by a transthoracic approach. The child has been entirely well during the 2½ years since his operation (fig. 1a and b).

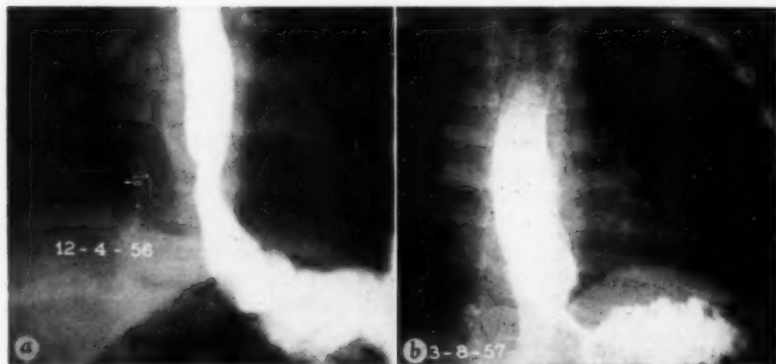


FIGURE 1a: Sliding-type diaphragmatic hernia: 2 or 3 cm. of the stomach extends above the hiatus in a 10-month-old boy. 1b: Esophagogastric junction is in the normal position 3 months after surgical reduction.

**Short Esophagus With Intrathoracic Stomach.** — Seventeen children had evidence of a short esophagus, as demonstrated by roentgenographic or esophagoscopic examination or as proved at the time of operation or necropsy. In some of these patients the reason for the short esophagus was evident but in others it was rather difficult to describe the exact sequence of events which led to the clinical picture seen at the time of our examination. Some of the more representative cases of short esophagus in this group will be described.

**Case 1:** A 5-year-old girl was registered at the clinic on February 20, 1950. It was recognized that she had congenital heart disease and the shadow of a large diaphragmatic hernia was noted in the posterior mediastinum. No treatment was recommended for either condition at that time. She returned in April, 1956, with cardiac failure. She was brought into reasonably good condition after digitalization and supportive therapy, and it was felt that further diagnostic procedures should be carried out. Accordingly, catheterization of the heart was done, but, unfortunately, the child died as a sequel to this procedure. Necropsy revealed pulmonary stenosis and cor biloculare. The spleen was absent and a large hiatal hernia was found with at least half of the stomach in the thorax. The esophagus was unusually short and did not measure more than half of its usual length. No evidence was found of esophagitis or obstruction.

Without doubt this case represents an instance of true congenital shortening of the esophagus. It illustrates the point that at times large hiatal hernias are completely asymptomatic, not only in adults but also in children. Furthermore, it calls attention to the work of Peters,<sup>7</sup> who stated that the congenital short esophagus is frequently associated with other congenital anomalies.

**Case 2:** A 3-year-old boy was seen a few weeks after he had swallowed lye and he remained under our observation until his death some 5 years later. Roentgenograms showed an excessively long cicatricial stricture of the esophagus (fig. 2). Dilatations were begun over a previously swallowed thread, and subsequent roentgenograms showed that the upper portion of the stomach had been pulled into the thorax. Over a 3-year period the strictured portion was dilated 37 times. Unfortunately, the results were not as favorable as those obtained in most patients with cicatricial strictures of the esophagus; finally total esophagectomy with anastomosis of the stomach to the hypopharynx was carried out. The results of this operation were never particularly satisfactory, and the child continued to have signs of obstruction at the anastomosis of the hypopharynx with the stomach. Further dilatations



**FIGURE 2:** Stricture caused by lye, with hiatal hernia: advanced irregular constriction and shortening of the distal half of the esophagus are evident with about 2 or 3 cm. of the stomach above the diaphragm.

were carried out with the use of a previously swallowed thread as a guide. Intussusception finally developed which in turn was related to a mass of thread previously swallowed to aid in the dilatations. Death occurred after an emergency operation.

In this case it was felt that the presence of intrathoracic stomach was a factor in the rapid recurrence of esophageal obstruction after dilatation. The regurgitation of acid gastric secretions into the esophagus was probably responsible for the constant esophagitis.

**Short Esophagus With Early Obstructive Symptoms.** — Four of the 17 children with short esophagus had difficulty in swallowing from early life. All four cases are described briefly.

**Case 3:** A 1-month-old girl was seen with what appeared to be stenosis of the esophagus. No evidence of a tracheo-esophageal communication could be demonstrated. Numerous attempts were made to treat this child by means of dilatation but much difficulty was encountered. On two occasions the child suffered an esophageal perforation requiring drainage of the pleural space. Finally, it was decided to attempt esophagectomy, but, unfortunately, the child died after this procedure. Roentgenograms of the esophagus made at the time of the child's first visit and 21 months later, just before operation, are shown in figure 3a and b.

**Case 4:** A girl seen about 1 month after birth because of esophageal obstruction was found at esophagoscopy to have a smooth stricture without evidence of esophagitis. A total of 11 dilatations were done over a previously swallowed thread during a 2-year period. Follow-up study shows that she has done extremely well and has not required any further treatment (fig. 4a and b).

**Case 5:** A 2-year-old boy had been unable to swallow anything but liquids from the age of 3 months. Efforts to give him solid foods were unsuccessful and he was found to have a long stricture of the esophagus with dilatation of the proximal end. This child has been treated by means of dilatation over a previously swallowed thread for 11 years. During this time he has had 19 dilatations, and although roentgenograms still show evidence of considerable obstruction he gets along remarkably well.

**Case 6:** A 2-year-old boy who was seen at the clinic had difficulty swallowing solid foods. In fact, no serious effort had been made to feed the child solid food until he was 10 months of age. At esophagoscopy he was found to have a smooth stricture. Eight dilatations have been done over a 3-year period and he has responded well.

Each of the four children in cases 3, 4, 5 and 6 had long stenoses of the esophagus associated with intrathoracic stomach. The history in

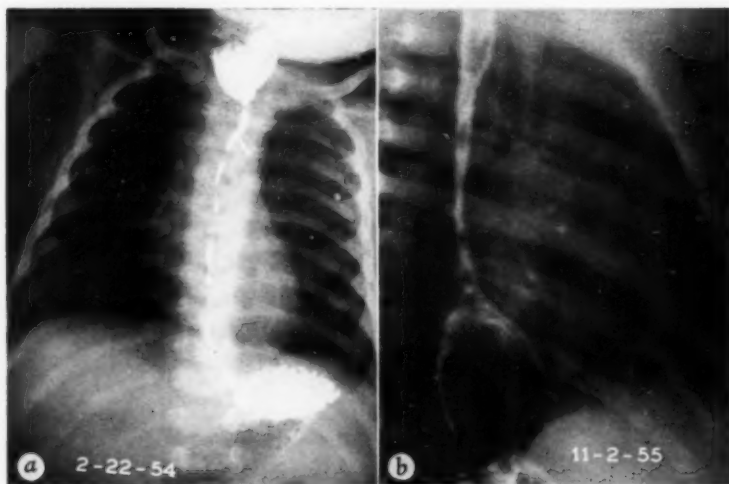


FIGURE 3a: Stenotic esophagus: long, irregular, narrow lumen of the esophagus with high-grade obstruction in a 1-month-old infant. 3b: Examination 21 months later showed short stenotic esophagus and a portion of the stomach above the diaphragm.

each case would suggest that the patient was born with stenosis of the esophagus. The hiatal hernia was either present at birth or developed after the dilatations.

**Short Esophagus With Stricture.** Initial History of Postural Regurgitation. — The histories of nine patients suggested incompetence of the lower sphincter of the esophagus<sup>3</sup> or chalasias. In each case the parents of the patient suggested that regurgitation was related to posture and, therefore, it is entirely possible that the esophageal stricture developed as the result of prolonged esophagitis. The actual onset of obstructive symptoms varied from 3 months to 4 or 5 years, but insofar as we could tell, regurgitation had begun at birth. Brief reports of three representative cases follow.

**Case 7:** A 4-year-old boy was registered at the clinic on June 21, 1941. He had had postural regurgitation from birth but no particular difficulty in swallowing had developed until shortly before his registration. At esophagoscopy, a smooth stricture was found without evidence of esophagitis. Dilatations were begun over a previously swallowed thread. Subsequent treatment was continued with the co-operation of his home physician. Roentgenograms of the esophagus taken 9 years apart are shown in figure 5a and b. This boy has done extremely well, and no dilatations have been required in the last 5 years.

**Case 8:** A girl, first seen at the clinic in July, 1947, at the age of 8 years, had had symptoms of postural regurgitation from early infancy and obstructive symptoms had developed later. Some dilatations had been done before we saw her. Roentgenograms showed a stricture in the lower third of the esophagus. A series of 24 dilatations was given over a 9-year period. The mother wrote that the patient has been exceptionally well and has not required any dilatation since we last saw her in 1956.

**Case 9:** A 5-year-old boy was registered at the clinic in 1950. He had a history of postural regurgitation since birth and had had hematemesis at the age of 1 year. All obstructive symptoms had developed within a year. We carried out dilatations on two occasions and subsequent dilatations were performed in his home community during the next 7 years. The results, however, were not satisfactory, and resection of the esophagus with interposition of the right colon was performed by surgeons in his home community in 1957.<sup>4</sup> The results of this operation have been excellent.

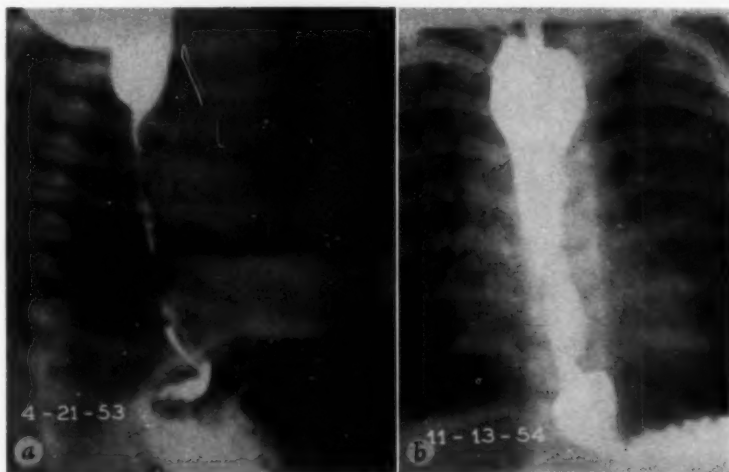


FIGURE 4a: Stenotic esophagus: long, smooth, narrow lumen of the esophagus with some obstruction in a 1-month-old infant. 4b: Appearance after treatment by dilation: increase in caliber of stenotic segment and small esophageal hiatal hernia 19 months later.

The histories of the nine patients in this group suggest that in each case stricture was the result of peptic esophagitis and the subsequent cicatricial changes associated with healing. Six of these patients have been treated satisfactorily by means of periodic dilatations over a previously swallowed thread. One child, as mentioned, was treated elsewhere by means of a colon transplant. Two patients have been lost to follow-up.

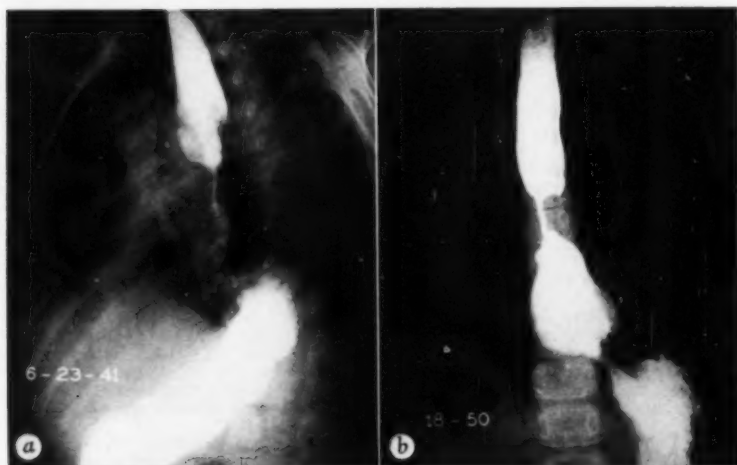


FIGURE 5a: Stricture of the esophagus: short, narrow segment below which there is a long supradiaphragmatic portion of the stomach in a 4-year-old boy. 5b: Appearance 9 years later, during which period dilations were carried out.



FIGURE 6: Hiatal hernia with ulceration in the terminal portion of short esophagus.

**Short Esophagus With Stricture.** Symptoms Occurring Late in Childhood. — In two children in our series no symptoms developed until they were several years old. Both cases are described.

**Case 10:** A 7-year-old boy registered at the clinic on October 9, 1956. At the age of 4 weeks this child had had projectile vomiting of blood. Abdominal operations were performed on two occasions a week apart, and apparently no cause for the bleeding was found. The exact findings at the time of operation are not known, but apparently the child had no further difficulty until he was 4 years of age, at which time he swallowed a coin which stuck in his esophagus and an emergency esophagoscopy was required. Dysphagia began at the age of 5, and at that time dilatations were carried out. From a historical standpoint, the sequence of events is difficult to analyze. However, we found a hiatal hernia with an apparent short esophagus and an ulcer at the esophagogastric junction (fig. 6). At esophagoscopy the ulcer could be demonstrated, as well as some associated esophagitis. Although stenosis was not demonstrated at the time of esophagoscopy, the child subsequently required dilatation. He has apparently done rather well on medical management alone, according to recent information from his family.

**Case 11:** A 14-year-old boy registered at the clinic on December 10, 1952, who apparently had not had esophageal symptoms until the age of 8 years, when obstructive symptoms began. Roentgenograms showed partial obstruction of the midesophagus with a peculiar beaded appearance (fig. 7a). A number of dilatations have been carried out (fig. 7b) but the results were not long lasting. Esophagoscopy demonstrated considerable evidence of esophagitis above the stricture. In the hope of controlling the problem of gastric acid and making treatment more effective, vagotomy and pyloroplasty were done in August, 1958, and further dilatations have been carried out with considerably greater success.

The etiology of the condition in cases 10 and 11 is far from clear. However, they do differ from the other cases in that symptoms developed at a relatively late stage. The patient in case 10 is the only one in our series who had a well-defined peptic ulcer at the esophagogastric junction. One wonders if this lesion falls into the category of the so-called Barrett ulcer. In both of these cases we are reminded of the so-called lower esophagus lined by columnar epithelium which was described by Barrett<sup>3</sup> and likewise considered by Allison and Johnstone.<sup>4</sup>

**Additional Comment Concerning Case Presentations.** — Although no conclusions can be drawn concerning paraesophageal hernias in children on the basis of our two cases, it would certainly seem reasonable



FIGURE 7a: Midesophageal partial obstruction with dilation above and "beaded" appearance or narrow segment and associated herniation of part of the stomach. 7b: Considerable change in appearance immediately after dilation. Irregularity of outline suggests presence of esophagitis.

to recommend that these hernias be repaired as soon as possible after the diagnosis has been made. Often the presenting symptoms are those of incarceration<sup>7</sup>, and definitive treatment is mandatory. As in adults, paraesophageal hernias often do not present the problem of incompetence of the cardia with reflux esophagitis.

On the other hand, patients with sliding hernias usually present the picture of incompetent cardia. In infants and small children obviously it is difficult to elicit the symptom "heartburn," but often postural regurgitation is evident and the physician should suspect the presence of a sliding hernia with incompetence of the cardia. Hematemesis should suggest the possibility of peptic esophagitis. Hiatal hernia usually can be demonstrated by the roentgenologist, even in small infants. At esophagoscopy, esophagitis can be detected and the length of the esophagus can be estimated. Surgical repair may be indicated in most infants and children with sliding hernias<sup>8,9</sup>, and the presence of esophagitis would represent a strong indication for surgical intervention. In our series we saw only one patient with sliding hernia before shortening had taken place.

With the exception of the asymptomatic hiatal hernia and short esophagus occurring in the patient with congenital heart disease and the hiatal hernia associated with the stricture caused by lye, it was most difficult to be positive about the actual cause of the short esophagus in any of the patients. In four patients we have reason to believe that stenosis of the esophagus was the primary disorder. In the remaining cases it would certainly seem that the stricture was a secondary phenomenon and apparently a result of regurgitation esophagitis. Whether the initial disorder was congenital hiatal hernia, congenital short esophagus, chalasias, or "lower esophagus lined by columnar epithelium," as suggested in two cases, it is indeed difficult to say.

In our hands, treatment of the short esophagus with stricture in children has been conservative. In the two instances in which we used esophagogastric anastomosis, the results were not favorable. For the most part, dilatation with a previously swallowed thread as a guide has proved to be reasonably satisfactory. Ten of our patients treated by dilatation and conservative methods are known to be doing exceptionally well or are being satisfactorily managed. Two children have been lost to follow-up. One child has had vagotomy and pyloroplasty in the hope of making his conservative treatment more satisfactory. Another one has had a successful operation performed elsewhere, in which the colon was transplanted to the thorax as a prosthesis.<sup>4</sup>

### Discussion

Much progress has been made toward understanding the various factors involved in the development of esophageal hiatal hernias in adults.<sup>2,8-11</sup> A brief review of some of our information on this subject will be helpful in pointing out how the problem of hiatal hernia in adults differs from that in children.

It is well recognized that the esophageal hiatus of the diaphragm is formed by the right crus which divides and encircles the orifice through which the esophagus passes into the abdomen. Of great concern are the anatomic and physiologic factors which tend to keep the esophagogastric junction in its normal position and which, in effect, prevent herniation of the stomach into the thorax. The afore-mentioned crural fibers of the diaphragm are said to form a pinchcock, sometimes called a "sling," which, by its action, tends to anchor the cardia in its normal position. Furthermore, a fibrous structure called the "diaphragmatico-esophageal ligament" or membrane has been

described which likewise tends to hold the junction in its proper place. In addition, some authorities<sup>4</sup> have stressed the importance of the left gastric artery and the vagus nerves which tend to hold the stomach in its normal position below the diaphragm. In addition to such anatomic factors, a scissorslike effect exercised by the crural fibers surrounding the hiatus has been described during inspiration and expiration,<sup>2,12</sup> which provides a physiologic explanation for a stable relationship between the esophagogastric junction and the hiatus.

The occurrence of esophageal hiatal hernias of the sliding type in adult patients has been explained by the demonstration of degenerative changes in the aforementioned anatomic structures.<sup>13</sup> The diaphragmatico-esophageal ligament becomes attenuated in older people; in fact, this structure becomes so frail that Barrett has said, "the membrane must be dissected out with eye of faith." It has been shown that there is a thinning of the crural muscles of the diaphragm and a loss of the elastic fibers, thus providing for greater mobility of the lower portion of the esophagus. However, the most important factor in the development of hiatal hernias seems to be an increase in intra-abdominal pressure. Such factors as obesity, pregnancy and the use of abdominal corsets and belts may be responsible for an increase in pressure. Furthermore, it has been stated that sudden compression of the abdomen as the result of trauma may result in widening the already weakened esophageal hiatus. In addition, it must be remembered that intra-abdominal pressures invariably exceed the pressures within the thoracic cavity, and, in view of these circumstances, it would seem remarkable that hiatal hernias do not occur more frequently.

Recently Botha<sup>13</sup> has made a detailed study of the esophageal hiatus of the diaphragm in infants. He has shown that in the normal infant the phreno-esophageal ligament is not only a real structure but also a strong and heavy structure. In addition, the crural muscles which surround the hiatus are greatly thickened and produce a well-defined diaphragmatic "tunnel." These thick crural fibers are placed in such a way that this tunnel enters the abdomen in an oblique direction, a feature which is certainly less obvious in the adult.

Obviously, the mechanism of development of hiatal hernia in children must be different from that which has been described for the aging adult. Although it seems rather certain that the sliding type of hiatal hernia in adults is an acquired phenomenon, similar hernias in infants probably have a congenital basis. Possibly these children are born with a large or malformed hiatus, and there is little to prevent herniation of the stomach from the abdomen into the thorax.<sup>3</sup>

In adults it is now generally recognized that the great majority of hiatal hernias of the short esophagus type have an acquired origin.<sup>12</sup> In most cases they develop from hiatal hernias of the sliding type. Although we have abundant physiologic proof that an effective lower esophageal sphincter exists (both in adults<sup>14</sup> and in children),<sup>15</sup> this sphincter often becomes incompetent when it is displaced into the thorax.<sup>16</sup> Regurgitation of gastric secretions into the esophagus frequently takes place in sliding hernias and produces peptic esophagitis; with alternate healing and ulceration, cicatrization and shortening of the esophagus take place. Regurgitant esophagitis will occur when the lower sphincter is destroyed by surgical procedures or occasionally when it is overstretched during dilatation for achalasia. Persistent vomiting may produce ulcerative esophagitis. In fact, any cause of esophagitis, chemical or inflammatory, may result in shortening and cicatrization of the esophagus, and thus a portion of the stomach is pulled into the thorax.

The explanation for short esophagus in children is not so clearly understood. It seems probable that in most instances the actual shortening of the esophagus is an acquired phenomenon just as it is in adults.<sup>9,12</sup> Carre and Astley<sup>17</sup> have shown that normal infants have an effective lower sphincter. However, pediatricians recognize the syndrome of chalasia or postural regurgitation<sup>3</sup> as a condition which, fortunately, corrects itself in most instances.<sup>8</sup> The majority of authorities feel that incompetence of the cardia is most uncommon except when associated with hiatal hernia.<sup>9,12,18,19</sup> In children, a congenital hiatal hernia is the best explanation for incompetence of the cardia, reflux esophagitis and subsequent stricture.

The true congenital short esophagus certainly exists, although it is undoubtedly rare.<sup>9,12,13</sup> Peters<sup>2</sup> said that it is often associated with other congenital abnormalities, and that both the hernia and the short esophagus may be completely asymptomatic. Such a case was present in our series.

It also seems probable that some infants are born with congenital stenosis of the esophagus. In some instances, intrathoracic stomach may not be suspected or, for that matter, may not actually be present at birth, but, as the child grows, a portion of the stomach is drawn into the thorax. It is often difficult to distinguish these children from those born with hiatal hernia in whom an acquired stricture with shortening has developed.

Another type of anomaly must be mentioned, namely, the lower esophagus lined by glandular epithelium. Barrett<sup>4</sup> described this esophagus as normal in all respects except for the nature of its mucosal lining. Because the patients have secreting glandular mucosa above the lower esophageal sphincter, esophagitis is likely to develop and in some patients a solitary ulcer, which has been labeled as a "Barrett ulcer," develops at or below the esophagogastric junction.

To complete the picture, one should mention the short esophagus that is the result of esophagitis and cicatrization from the ingestion of caustics such as lye, or that may be produced by intense vomiting. Not infrequently, some of the stomach is drawn into the thorax in conjunction with such unfortunate occurrences.

When a definite diagnosis of hiatal hernia of either the paraesophageal or sliding type can be made in an infant or child, there is certainly every reason to consider repair of the lesion. In paraesophageal hernia, the surgeon is able to relieve obstruction and bring about normal anatomic relationship. In sliding hernia, the surgeon should be able to restore the competence of the lower sphincter of the esophagus and thus prevent the development of regurgitation esophagitis with all of its complications. In our cases of short esophagus with stricture it is hard to know how many of these strictures might have been prevented had early repair of the sliding hernia been carried out.

The problem of therapy for the short esophagus with stricture is indeed difficult. It is impossible to repair these hernias by any conventional means. Resection of the stricture with high esophagogastrectomy has been recommended by Husfeldt and associates,<sup>9</sup> and certainly there are instances in which satisfactory results have been obtained. The problems that are likely to arise after esophagogastrectomy are often serious, and surgeons have been looking for a better solution to the problem. Recently efforts have been made to interpose a segment of intestine or to bring the right colon into the thorax. These procedures have met with varying degrees of success. In a child, the matter of further growth is of some importance, and it is often difficult to know whether the surgically inserted prosthesis will grow at the same rate that the child does. Heretofore it has been our policy to be as conservative as possible in the management of these conditions.

We have employed a technic of dilation by which a previously swallowed thread is used as a guide.<sup>17</sup> In small children dilations are usually done after the administration of an anesthetic. Older children, however, are usually able to tolerate dilations satisfactorily without anesthesia. A flexible wire spiral, graduated Plummer sounds and a whalebone are needed for the dilations. The thread is passed through the spiral and in turn guides the dilating instrument through the stricture into the stomach. The size of the sounds varies, of course, depending on the size of the child and the degree of obstruction. Often it is necessary to begin with No. 20 to 22 F. sounds and to carry out periodic dilations over a period of weeks, months and years.

In the early stages of treatment, dilations are usually performed about once a week, but as the size of the sounds is increased the interval between dilations becomes greater. As the larger sounds are passed, relief from dysphagia lasts longer and soon 3 to 6 months may elapse between treatments. Ultimately the stricture is dilated to the size of a No. 45 F. sound, but even after reaching this goal, dilations probably should be repeated annually.

The problem of incompetence of the cardia is certainly not solved by dilating the stricture. If anything, regurgitation occurs even more readily after dilation. Therefore, it is necessary to continue medical treatment. It is always desirable that the children sleep with the head at a higher level than the feet; this is usually accomplished by placing wooden blocks 4 to 8 inches high under the head of the patient's bed. In addition, antacid medication should be taken regularly by the patient, shortly after meals and especially at bedtime. Constricting abdominal garments should not be worn.

Admittedly, the results of therapy by dilation are not ideal. However, we have found the results to be generally satisfactory, and it is our feeling that conservative methods should be tried and that surgical resection of the esophagus should be deferred whenever possible. When the child has reached his full growth the question of surgical resection can be reconsidered. However, most of our patients have done so well with conservative management that surgical treatment has not been necessary.

#### SUMMARY

Hiatal hernias are rare in infants and children, as evidenced by the fact that only 20 patients less than 15 years of age with this condition have been seen at the Mayo Clinic in the last 9 years. By contrast, several thousand adults with hiatal hernias have been seen during the same period. Most of the adults were in the older age group.

Of the 20 hiatal hernias encountered in children, two were of the paraesophageal type, one was a sliding hernia, and the remainder were of the short esophagus type. We suspect that in most instances hiatal hernias have a congenital basis in children, but shortening of the esophagus may or may not be an acquired phenomenon.

Hiatal hernias of the paraesophageal and sliding type should be repaired as soon as a diagnosis can be made. Most of our patients with short esophagus came to us with strictures. Treatment in these cases invariably presents problems and the usual surgical methods of repair for hiatal hernia cannot be adapted in cases of short esophagus.

In most of our patients with short esophagus and stricture, a conservative program consisting of dilations over a previously swallowed thread and medical measures such as routine use of antacids and elevation of the head of the patient's bed has been

employed. Satisfactory results have been obtained in two thirds of the patients treated by conservative means.

### RESUMEN

Las hernias hiatales son raras en infantes y en niños según lo revela el hecho de que sólo 20 enfermos menores de 15 años de edad se han visto con esta afección en la Clínica Mayo durante los últimos 9 años. Por el contrario, varios millares de adultos se han visto con hernias hiatales durante ese período. La mayoría de los adultos se encontraban entre los de mayor de edad.

De las 20 hernias hiatales encontradas en niños, dos fueron del tipo paraesofágico, una fué de deslizamiento y el resto del tipo de esófago corto. Sospechamos que en la mayoría de los casos las hernias hiatales tienen una base congénita en los niños, pero el acortamiento de esófago puede ser o no un cambio adquirido.

Las hernias hiatales de los tipos paraesofágico y de deslizamiento deben ser reparadas tan pronto como se haga el diagnóstico.

La mayoría de los enfermos con esófago corto, llegó a nosotros con estenosis. El tratamiento en estos casos presenta invariablemente problemas y los métodos habituales para la reparación de la hernia hiatal no pueden adaptarse a los casos de esófago corto.

En la mayoría de nuestros enfermos con esófago corto y estenosis, se sigue un plan conservador que consiste en dilataciones siguiendo a un hilo deglutido y a medidas médicas tales como el uso habitual de antiácidos y elevación de la cabecera del enfermo. Se han observado resultados satisfactorios en dos tercios de los enfermos tratados conservadoramente.

### RESUMÉ

Les hernias hiatales sont rares chez les nouveau-nés et les enfants. Il est démontré qu'il n'y a eu que 20 malades âgés de moins de 15 ans atteints de cette affection à la Clinique Mayo dans ces neuf dernières années. Au contraire, plusieurs milliers d'adultes atteints de hernies hiatales ont été vus pendant la même période. La plupart des adultes étaient des gens déjà âgés.

Sur les 20 hernies hiatales rencontrées chez les enfants deux furent du type para-oesophagien, une fut une hernia par glissement et les autres furent celles qui appartiennent aux cas d'oesophage court. Les auteurs estiment que dans la plupart des cas, les hernies hiatales ont une origine congénitale chez les enfants, mais le raccourcissement de l'oesophage peut être ou ne pas être un phénomène acquis.

Les hernies hiatales du type para-oesophagien et du type par glissement devraient être traitées dès que le diagnostic en peut être fait. La plupart des malades porteurs d'oesophage court avaient également un rétrécissement. Le traitement dans ces cas pose invariablement des problèmes et les méthodes chirurgicales habituelles de correction des hernies hiatales ne peuvent être adaptées aux cas d'oesophage court.

Chez la plupart des malades porteurs d'oesophage court et de rétrécissement, les auteurs ont utilisé un traitement conservateur consistant en dilatations sur un fil avalé auparavant, et des moyens médicaux tels que l'utilisation systématique d'anti-acides et l'élévation de la tête du lit du malade. Des résultats satisfaisants ont été obtenus dans deux tiers des cas traités par ces moyens conservateurs.

### ZUSAMMENFASSUNG

Hiatus-Hernien sind selten bei Säuglingen und Kindern, wie sich aus der Tatsache ergibt, dass die Mayo-Klinik nur 20 Kranke unter 15 Jahren mit dieser Affektion in den letzten 9 Jahren aufnahm. Im Gegensatz dazu wurden mehrere tausend Erwachsene mit Hiatus-Hernien während der gleichen Zeitspanne untersucht. Die meisten Erwachsenen befanden sich im höheren Alter.

Unter den 20 kindlichen Hiatus-Hernien waren zwei vom paraoesophagealen Typ, einer mit einer Gleithernie und die übrigen sämtlich mit verkürztem Oesophagus. Wir neigen zu der Annahme, dass die Hiatus-Hernie in den meisten Fällen bei Kindern auf angeborener Grundlage beruht, wo hingegen die Verkürzung der Speiseröhre je nach dem eine erworbene Erscheinung sein kann, oder nicht.

Hiatus-Hernien vom paraoesophagealen und vom Gleit-Typus müssen beseitigt werden, sobald die Diagnose gestellt ist. Die meisten unserer Kranken mit verkürztem Oesophagus kamen zu uns mit Strikturen. Die Behandlung in diesen Fällen stößt jedesmal auf Schwierigkeiten, im die üblichen chirurgischen Methoden der Beseitigung von Hiatus-Hernien lassen sich auf die Fälle von Speiseröhrenverkürzung nicht anwenden.

Bei den Meisten unserer Kranken mit Oesophagusverkürzung und Striktur wurde ein konservativer Behandlungsweg eingeschlagen in Form von Dilatationen mit Hilfe eines vorher verschluckten Fadens und interne Massnahmen, wie etwa regelmässiger Gebrauch von säurebindenden Mitteln und Hochstellung des Kopfendes des Krankenbettes. Befriedigende Ergebnisse erzielten wir in zwei Drittel der Patienten, die mit konservativen Mitteln behandelt worden waren.

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# Resectional Therapy for Pulmonary Tuberculosis at Sunmount, 1950-1957, 807 Cases

## IV. Pulmonary Resection for Tuberculosis without Effective Chemotherapy

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In reviewing resectional therapy for pulmonary tuberculosis at Sunmount, it seemed of particular interest to evaluate those patients who had had resections without effective chemotherapy coverage.

A drug is considered to be ineffective, or the patient resistant to the drug, if the tubercle bacilli cultured from the patient grew in the following concentrations of the following drugs: streptomycin (SM), growth in 10 micrograms per ml.; isoniazid (INH), growth in one microgram per ml.; para-aminosalicylic acid (PAS), more than 50 colonies in 10 micrograms per ml.; viomycin (VIO), growth in 10 micrograms per ml. While it is realized that there are gradations in resistance of organisms, it is felt that the above definition has proven sufficiently useful clinically in this hospital to allow it to be used as a criterion for managing the tuberculous patient about to undergo resectional therapy.

In order for a patient to be classified as receiving ineffective chemotherapy, it was necessary for all the drugs that he was receiving to fall into the category of ineffectiveness by the above definition. Patients fell into the group of ineffective chemotherapy for two reasons:

1. No substitute chemotherapy was available. Since the time interval covered by the resection study spans the time interval from 1950 to 1957, a large percentage of patients in the ineffective chemotherapy group were operated upon in the early fifties.

2. When the specimen bacteriology showed more resistance than the last available pre-operative study which may have been considered susceptible, and if the specimen bacteriology fell into the range of ineffectiveness, as defined above, the patient was also considered to be receiving ineffective chemotherapy.

With this definition of ineffective chemotherapy, the group of patients studied has been divided into the four sub-groups of open-positive, closed-positive, open-negative, and closed-negative as defined by Decker et al.<sup>1</sup>

### *Open-Positive Cases*

Twenty-eight patients who were considered open-positive at the time of resection fall into the group of ineffective chemotherapy coverage. They are compared with 56 other cases picked at random, 28 of which received good drug coverage, or what is called Grade A in Table 1, and the other 28 received less effective chemotherapy called Grade B in Table 1. Of the patients with Grade A chemotherapy eight were receiving SM and PAS, nine SM and INH, five INH and PAS, two triple chemotherapy, and four INH and pyrazinamide (PZA). Grade B chemotherapy covers a variety of less effective combinations. Even disregarding Terramycin,

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TABLE 1 — OPEN-POSITIVE CASES  
Evaluation of Different Types of Chemotherapy for Resection

Drug Coverage	No. of Cases	No. with Tplsty	Major Compl.	Maj. Comp. in Tplsty. Group	Persistent Pos. Sputum	Late Relapses
Ineffective	28	14	13	7	11	4
Grade B*	28	7	2	1	1	2
Grade A*	28	5	0	0	0	0

\*See text for definition.

which was used in combination with INH in one case, and with viomycin on two occasions, eight different types of chemotherapy combinations are represented in this group of 28 patients. Eleven were on INH alone, seven on VIO and PZA, two on SM alone, one on only PAS, one on PAS and VIO, three on VIO alone, two on only PZA, and one on INH and VIO. Additional chemotherapy in this group, if considered ineffective by susceptibility studies as defined, is not listed. These different types of chemotherapy coverage are obviously not comparable, though arbitrarily included in the Group B group.

The results of surgery in these three groups of patients with varying chemotherapy is summarized in Table 1. Under major complications listed in Table 1 is meant either fistula, empyema, fistula and empyema, or occult fistula. Under persistent positive sputum is meant more than one positive sputum within three months following surgery.

The most striking finding in Table 1 is the very high major complication rate in the ineffective chemotherapy group. Almost 50 percent of the patients in this group had major complications. The presence of thoracoplasty does not seem to influence the complication rate as indicated in columns three and five in Table 1. By contrast, the major complication rate with good drug coverage was zero and with less effective chemotherapy, 7 per cent. The other striking finding in the ineffective chemotherapy group is the high incidence of persistent positive sputum following resection for cavitary disease. The relapse rate of these patients is also higher than in the patients with good chemotherapy.

In Table 2 it is pointed out that the extent of resection as carried out in the three groups of cases listed in Table 1 is roughly comparable as indicated by the number of lobes, segments, and wedges resected in the three groups. The incidence of reconstitution<sup>1</sup> in segmental resections is slightly higher in the group receiving better chemotherapy, and this may possibly have some bearing on the major complication rate. It is of interest to note that in the group with ineffective chemotherapy, segmental resections constituted part of the operation in 13 of the 28 cases. Eight of these developed a major complication. Of the five patients in whom segmental resection constituted part of the operation, but did not

TABLE 2 — OPEN-POSITIVE CASES  
Comparative Data on Extent of Resections, Reconstitution, and Specimen Bacteriology

Drug Coverage	Number of Cases	Lobe	Resections include:		Reconstitution	Positive Specimen
			Seg.	Wedge		
Ineffective	28	18	11	8	2	26
Grade B	28	17	12	8	4	24
Grade A	28	19	11	9	6	23

TABLE 3 — OPEN-POSITIVE CASES

Last follow-up status of patients who had resections with ineffective drug coverage.

1 yr.	2 yrs.	Inactive 3 yrs.	4 yrs.	5 yrs.	Active	No follow- up	Dead	Total
4	5	5	0	1	1	6	6	28

develop a major complication, two had reconstitution of raw lung surfaces. The problem of the importance of reconstitution has been discussed at length elsewhere.<sup>2</sup>

In Table 3, the present status of the twenty-eight open-positive cases who had resection without effective drug coverage is summarized. Fifteen of the twenty-eight patients are presently inactive. Additional chemotherapy of a different nature than that used for resection was usually necessary to achieve the state of inactivity. One patient is still active after five years. Six patients have been lost to follow-up, and six are dead. Of the six patients who died, the cause was progressive tuberculosis in two, erosion of an occult fistula into a major pulmonary vessel with massive hemorrhage in another two, and causes unrelated to tuberculosis in the remaining two patients. (One died of a cerebro-vascular accident after being inactive for three and a half years, and the other patient succumbed to acute alcoholism).

In Table 4 the present status of those patients in the open-positive group with ineffective chemotherapy who were persistently positive following surgery or who have had relapses is summarized. It is mainly of interest to note that with further surgery or additional chemotherapy a substantial percentage of these patients has become inactive. An almost equally impressive percentage has died as a result of tuberculosis.

#### Closed-Positive Cases

Seven patients with the pre-operative classification of closed-positive underwent resectional therapy. This small group comprises many patients who after a long period of negative sputum had an isolated positive culture just prior to surgery. Sometimes the classification of closed-positive had to be made in retrospect only after a pre-operative sputum culture report became available following surgery. The absence of any serious complications in this small group is notable, and is briefly sum-

TABLE 4 — OPEN-POSITIVE CASES

Last follow-up status of patients with persistent positive sputum and relapse who had resections with ineffective drug coverage.

	Number of cases	Inactive	Active	Turned negative	No follow- up	Dead
Persistent	11	2	1	2	3	3
Positive						
Relapses	4	2	0	0	1	1

TABLE 5 — CLOSED-POSITIVE CASES

Patients who had resections with ineffective drug coverage.

Number of cases	Number with Thoracoplasty	Major complications	Persistent positive	Relapse
7	0	0	0	0

TABLE 6 — CLOSED-POSITIVE CASES  
Last follow-up status

1 yr.	2 yrs.	Inactive:	4 yrs.	5 yrs.
		3 yrs.		
2	1	3	0	1

marized in Table 5. The present status of the closed-positive cases is presented in Table 6. All are presently inactive for varying periods of time.

#### *Open-Negative Cases*

Twenty patients with the pre-operative classification of open-negative had resectional therapy under ineffective drug coverage. Pertinent data of these patients is briefly summarized in Table 7. It is of interest that there was no major complication. More striking is the fact that there were six relapses, or a relapse rate of 30 per cent. This relapse rate is ten times greater than that observed in the group of operated open-negative cases previously reported from this hospital.<sup>1</sup> There is no relationship of relapses to the bacteriology of the resected specimens, as judged by culture or guinea-pig. Positive specimens were found in 50 per cent of all cases, and 50 per cent of the relapses. The present status of the open-negative cases is summarized in Table 8. With additional treatment 85 per cent are now known to be inactive. Only one case is known to have active tuberculosis, and two cases have been lost to follow-up.

#### *Closed-Negative Cases*

Twenty-six cases classified as closed-negative had resection under ineffective drug coverage. The pertinent data are briefly summarized in Table 9. There were two major complications, one tuberculous and one non-tuberculous. The present status of the closed negative cases is summarized in Table 10.

#### DISCUSSION

The importance of effective chemotherapy when undertaking resection for pulmonary tuberculosis is strikingly shown in the open-positive cases. In this group of patients, the major complication rate, relapse rate, and failure of conversion of sputum was forbiddingly high. In contrast, open-positive cases who had resections under good or even less than good chemotherapy coverage, as defined above, had excellent results as summarized in Table 1. In retrospect, it may be stated from these figures that they justify the definition of ineffective chemotherapy used at Sunmount, as defined in the introduction. The high relapse rate in the open-negative group also points out the need for an adequate period of post-resectional effective chemotherapy.

TABLE 7 — OPEN-NEGATIVE CASES  
Patients who had resections with ineffective drug coverage.

Number of cases	Number with Thoracoplasty	Major complications	Relapses
20	3	0	6

TABLE 8 — OPEN-NEGATIVE CASES  
Last follow-up status

1 yr.	3 yrs.	Inactive:	4 yrs.	5 yrs.	Active	Unknown	Total
		2 yrs.					
7	2	5	2	1	1	2	20

TABLE 9 — CLOSED-NEGATIVE CASES  
Patients who had resections with ineffective drug coverage.

Number of cases	Number with Thoracoplasty	Major complications	Relapses
26	1	2	1

TABLE 10 — CLOSED-NEGATIVE CASES  
Last follow-up status

1 yr.	2 yrs.	Inactive: 3 yrs.	4 yrs.	5 yrs.	Unknown	Dead	Total
4	5	4	8	0	4	1	26

The low complication rate in the closed-positive, closed-negative, and the open-negative groups is probably a reflection of host resistance and low quantitative or absent contamination with tubercle bacilli.

#### SUMMARY

The experience with resectional therapy under ineffective chemotherapy coverage for pulmonary tuberculosis has been reviewed. The definition of ineffective chemotherapy as presently used at Sunmount has been presented and the validity of this definition justified by the poor surgical results in the open-positive group. This study again emphasizes the importance of effective chemotherapy before and after resectional therapy for pulmonary tuberculosis.

#### RESUMEN

Se ha revisado la experiencia de tratamiento por resección bajo la protección de la quimioterapia inefectiva en tuberculosis pulmonar.

La definición de quimioterapia inefectiva tal como se usa en Sunmount se ha presentado y la validez de esta definición se justifica por los deficientes resultados obtenidos en el grupo de positivos abiertos. Este estudio otra vez recalca la importancia de la quimioterapia efectiva antes de la resección en tuberculosis pulmonar.

#### RESUMÉ

L'auteur a passé en revue l'expérience de la thérapeutique chirurgicale quand la chimiothérapie de la tuberculose pulmonaire se révéla inefficace. La définition de la chimiothérapie inefficace, telle qu'on la comprend actuellement à Sunmount, est exposée, et la valeur de cette définition justifiée par les médiocres résultats de la chirurgie dans le groupe de malades atteints de tuberculose active avec expectoration positive. Cette étude souligne de nouveau l'importance d'une chimiothérapie efficace avant et après traitement chirurgical pour tuberculose pulmonaire.

#### ZUSAMMENFASSUNG

Die Erfahrungen mit der Resektionstherapie bei Lungentuberkulose unter dem Schutz unwirksamer Chemotherapie wurden mitgeteilt sowie die Definition einer unwirksamen Chemotherapie, wie sie gegenwärtig in Sunmount-Hospital gebraucht wird. Die Stichhaltigkeit dieser Definition ist gerechtfertigt aufgrund der schlechten chirurgischen Ergebnisse in der Gruppe der offen-positiven Fälle. Diese Untersuchung betont von neuem die Wichtigkeit effektiver Chemotherapie vor und nach der Resektionstherapie wegen Lungentuberkulose.

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## Spontaneous Pneumothorax\*

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This study is a review of 39 consecutive cases of spontaneous pneumothorax comprising 44 episodes, treated at Westminster DVA Hospital, London, Ontario, for the period ending Dec. 31, 1958.

The treatment of spontaneous pneumothorax on the chest service of this hospital has been conservative, and consists of bed rest with aspiration of air if respiratory embarrassment is present or if there is no radiographic evidence of beginning re-expansion after 14 days. If respiratory embarrassment is present the intrapleural pressures are estimated in order to determine the presence or absence of tension pneumothorax. Hemopneumothorax from the time of admission is followed with close liaison between the departments of medicine and surgery. Treatment consists of replacement of blood loss and repeated aspiration of the pleural cavity until dry. Thoracotomy is recommended if clinical observation indicates that bleeding is progressive and uncontrolled.

The majority of patients gave no history of previous pulmonary disease (Table 1). An appreciable number demonstrated the chronic bronchitic syndrome and/or emphysema. The one case of tuberculosis had been quiescent for seven years.<sup>4,5,6</sup>

Table 2 shows the main presenting signs and symptoms. All patients presented with pain and/or dyspnea. The pain was pleuritic in character. Cyanosis was present in those with significant lung collapse. The degree of collapse and the severity of dyspnea were not always proportional.

Table 3 shows that there were four errors in clinical diagnosis without the aid of a chest radiograph. The patient diagnosed as myositis had minimal collapse of the lung. Two of the other three errors occurred in patients with hemopneumothorax.

Table 4 shows the radiographic findings as to the presence and extent of pleural effusion. The two cases of large effusion were proved hemo-

TABLE 1 — PREVIOUS PULMONARY HISTORY

None	27
Previous Spontaneous Pneumothorax	5
Chronic Bronchitis	12
Emphysema	6
Tuberculosis	1

TABLE 2 — PRESENTING SYMPTOMS

	Numbers
Pain Alone	11
Dyspnea Alone	3
Pain and Dyspnea	25
Cyanosis	7
Hemoptysis	0

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TABLE 3 — ADMISSION DIAGNOSIS

	Numbers
Pneumothorax	35
Lobar Pneumonia	1
Broncho Pneumonia	1
Pleural Effusion	1
Myositis	1

pneumothoraces as was one with a moderate effusion. The small effusions were those showing radiographic evidence of obliteration of the costophrenic angle on the involved side.

Table 5 consists of essential data derived from this series. The case requiring 279 days for re-expansion demands special comment and is detailed separately in brackets. This was in essence a patient with chronic pneumothorax who refused treatment. Repeat examination eight months later revealed complete re-expansion. It can therefore be seen that the time element here may be inaccurate.

Three cases of hemopneumothorax were encountered in the 18-40 age group; none of these required thoracotomy either for control of haemorrhage or decortication.

All cases requiring aspiration of air, which as previously stated was done because of respiratory embarrassment, had over 50 per cent lung collapse. The lungs re-expanded fully in all cases. No residual atelectasis or evidence of sepsis was demonstrated in any case.

The extent of collapse was estimated from the first radiographic examination of the chest. Fifty-six per cent of all patients, and 45 per cent of the patients treated by bed rest demonstrated more than 50 per cent collapse of their lungs.

The re-expansion time was computed from the date of onset of symptoms to the date of radiographic confirmation of complete re-expansion. The 29 patients treated by bed rest showed an average re-expansion time of 29 days.

The recurrence rate at Westminster Hospital has been approximately 10 per cent.

### Discussion

The introduction of a catheter with active suction is considered the method of treatment giving the most rapid re-expansion of the lung; from immediate re-expansion<sup>3</sup> to 3.5 days.<sup>4</sup>

Increased morbidity and the possible development of chronic pneumothorax are the arguments advanced against conservative therapy.

The points against more aggressive treatment are the possible development of broncho-pleural fistula and permanent anatomical damage resulting from various types of operative interference.

In recent articles, the majority of authors<sup>1,2,3,8,9,13</sup> advocate an aggressive approach in the treatment of spontaneous pneumothorax. Active therapy recommended varies from the use of catheter suction in all cases<sup>8</sup> to thoracotomy if the lung fails to re-expand after five to seven days of suction and if bronchoscopy is negative.<sup>8,9</sup> Reported results are difficult to interpret especially as regards morbidity in patients submitted

TABLE 4 — PRESENCE OF EFFUSION

None	24
Effusion: Slight	10
Moderate	3
Marked	2

TABLE 5 — SUMMATION OF DATA

<i>Bed Rest:</i> — Numbers:	21	2	6	29
Re-expansion Time:				
Days — Range	8-70	23-49	22-58	8-70
Average	25	36	41	29
<i>Air Aspiration:</i> — Numbers:	1	1	4	6
Re-expansion Time:				
Days — Range	31	108	23-60 (229)	23-108 (279)
Average	31	108	36 (97)	49 (88)
<i>Blood Aspiration:</i> — Numbers:	3	0	0	3
Re-expansion Time:				
Days — Range	23-93			23-93
Average	52			5
<i>Catheter Plus Water Seal:</i>	0	1	0	1
Re-expansion Time:				
Days — Range		7		7
Average				

to thoracotomy. It would seem reasonable to assume that duration of hospitalization would be considerably prolonged if cases subjected to thoracotomy were included.

Wynn Williams<sup>10</sup> and Cliff<sup>11</sup> discussing a conservative regime report good results, similar to our own.

To date the results on the medical service at Westminster Hospital compare favorably with the results reported elsewhere. We feel that the continuation of conservative management in the treatment of spontaneous pneumothorax is warranted.

#### SUMMARY

Thirty-nine consecutive cases of spontaneous pneumothorax treated with a conservative approach at Westminster DVA Hospital are analyzed.

1. No previous pulmonary history was given in 27 patients; the remainder gave a history in keeping with chronic bronchitis and/or emphysema.
2. Pain and/or dyspnea were initial symptoms in all.
3. Four were misdiagnosed on initial clinical examination prior to chest radiography.
4. Fifteen demonstrated an effusion on radiographic examination, three of which were obvious on clinical examination.
5. The average re-expansion time for 35 patients treated by needle aspiration and/or bed rest was 32 days.
6. The recurrence rate was 10.2 per cent.
7. No immediate or remote complication was encountered in this series.
8. We disagree with the early thoracotomy recommended by some authors.

#### RESUMEN

Se analizan treinta y nueve casos consecutivos de neumotórax espontáneo, tratados de manera conservadora en el Hospital DVA de Westminster.

1. En 27 enfermos no había antecedente alguno de enfermedad pulmonar, el resto dió una historia ya sea de bronquitis y/o enfisema.
2. Los síntomas iniciales fueron dolor y disnea o ambos.
3. Cuatro fueron erróneamente diagnosticados antes de la radiografía de tórax.
4. En quince había derrame al examen radiográfico, de los que evidentemente tres existían ya cuando se hizo el estudio clínico.
5. El término medio de tiempo que requirió la expansión para 35 enfermos tratados con aspiración por aguja y reposo en cama o sin éste, fué de 32 días.
6. La recurrencia fué de 10.2 por ciento.
7. No hubo complicaciones inmediatas o tardías en esta serie.
8. No estamos de acuerdo con la recomendación de algunos autores que haven la toracotomía temprana.

#### RESUMÉ

Les auteurs analysent 39 cas de pneumothorax spontanéés traités par des procédés conservateurs à l'Hôpital DVA de Westminster.

1. Il n'y avait aucun antécédent chez 27 malades; les autres avaient une histoire de bronchite chronique associée ou non à de l'emphysème.

2. La douleur associée ou non à la dyspnée furent les symptômes de début dans tous les cas.
3. Il y eut quatre erreurs de diagnostic lors de l'examen clinique initial avant la radiographie thoracique.
4. Chez 15 malades on constata un épanchement liquidien sur la radiographie, trois d'entre eux étaient évidents à l'examen clinique.
5. Le temps moyen de réexpansion pour les 35 malades traités par aspiration à l'aiguille associée ou non au repos au lit fut de 32 jours.
6. Le taux de rechute fut de 10.2%.
7. On ne rencontra pas de complication immédiate ou éloignée dans ce groupe.
8. Les auteurs désapprouvent la thoracotomie précoce conseillée par certains auteurs.

#### ZUSAMMENFASSUNG

Analyse von 39 aufeinander folgenden Fällen von Spontan-pneumothorax, die im Westminster DVA-Krankenhaus auf konservativem Wege behandelt wurden:

1. 27 Kranke gaben in der Vorgeschichte keine Lungenkrankheiten an; die übrigen berichteten übereinstimmend von chronischer Bronchitis und/oder Emphysem.
2. Schmerz/oder Dyspnoe waren in allen Fällen das initiale Symptom.
3. Bei 4 Fällen wurde eine Fehldiagnose gestellt bei der ersten klinischen Untersuchung vor der Thorax-Röntgenuntersuchung.
4. Fünfzehnmal war ein Erguß nachweisbar bei der röntgenologischen Untersuchung, wovon 3 bei der klinischen Untersuchung schon erkennbar waren.
5. Die durchschnittlich Wiederausdehnungszeit betrug bei 35 Kranken, die mit Nadel-Absaugung und/oder Bettruhe behandelt worden waren, 32 Tage.
6. Die Häufigkeit der Rückfälle lag bei 10.2%.
7. Bei dieser Beobachtungsreihe wurden keine unmittelbaren oder verborgenen Komplikationen festgestellt.
8. Wir pflichten der frühzeitigen Thorakotomie, wie sie von einigen Autoren empfohlen wird, nicht bei.

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## Does a Carcinomatous Scalene Node Contraindicate Pulmonary Resection?

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Following the recognition by Daniels,<sup>1</sup> and others<sup>2,3,4</sup> that lymphatic metastases to scalene nodes occur in bronchogenic carcinoma, some investigators have felt that this finding is an absolute contraindication to resection of the tumor. The conclusions drawn from the experience of Shields,<sup>5</sup> Skinner<sup>6</sup> and Harken<sup>7</sup> and their respective co-workers indicate a direct relationship between positive nodes and a poor prognosis. Others such as Shapiro and Palumbo,<sup>7</sup> and Cruze et al<sup>8</sup> place less significance in this finding. With the increased utilization of extended intrapericardial resection of heretofore non-resectable tumors, many cases can be resected in the presence of positive scalene nodes. However, this extended resection is not without an increased morbidity and mortality. This may range as high as 23 to 25 per cent as compared with 7.7 per cent for the standard type of pneumonectomy.<sup>9</sup> In contradistinction to carcinoma of the esophagus or colon, palliative resection of lung tumors imposes a significant physiologic insult in terms of diminished respiratory reserve, whereas palliative resection in the alimentary tract restores function. Pulmonary resection could, however, be justified by the prevention of pneumonitis, lung abscess or significant recurrent hemoptysis secondary to the tumor. Should these complications be of minor significance in those cases not resected when compared to those following distant metastases, pulmonary resection would not appear justified.

We have reviewed the cases of 25 patients with proved carcinoma of the lung, metastatic to the scalene lymph nodes, upon whom lung resection was not performed. The study covered patients seen from 1953 to the present time. Information was contributed by the referring physician and our own records concerning the length of survival, cause of death and complications of the terminal illness.<sup>†</sup>

### *Findings*

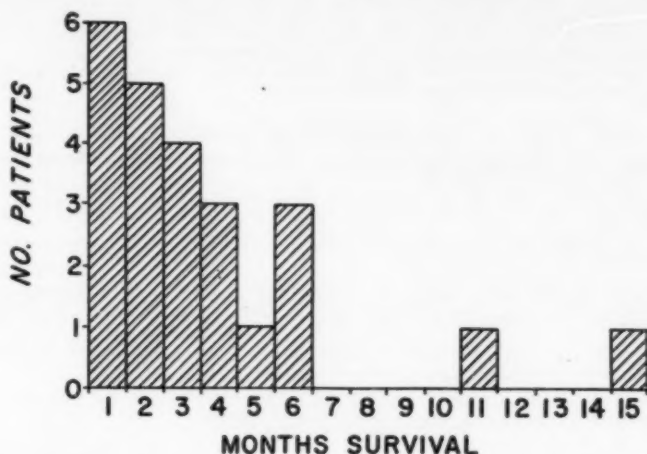
All 25 patients have expired by the time of preparing this manuscript. Twenty-four survived an aggregate of 89 months following biopsy, the range being from five days to 15 months with a mean survival of 3.6 months (Figure 1). Those that had the longest survival had undifferentiated carcinoma treated with irradiation. Two died of causes not related to malignancy. Six died with pneumonitis and six had significant sputum, although not enough to be a major problem. Only three had significant hemoptysis, but in no case did exsanguination or blood loss anemia result. Although 10 had severe dyspnea and 10 also had thoracic pain in the terminal stages, many had these same symptoms as their presenting complaints at the time of diagnosis. Four had extension of the tumor to the opposite lung. Hepatic metastases occurred in five, osseous

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†Biopsies were performed by Drs. L. D. Hill, R. Yore and G. H. Lawrence.

**BRONCHOGENIC CARCINOMA  
METASTATIC TO SCALENE LYMPH NODES  
(25 patients, 1 unreported)**



metastases in five, soft tissue metastases in five and brain metastases in four patients. All but four had some combination of two or more significant symptoms as well as some form of extra thoracic metastasis.

#### SUMMARY

Scalene node biopsy is of proved value in the diagnosis of pulmonary malignancy, and likewise has prognostic significance. The mean life expectancy in twenty-four cases following positive biopsy is 3.6 months with a majority surviving three months or less. The complication of the disease were often present at the time of diagnosis. Pulmonary complications were often overshadowed by extra pulmonary metastases. In no patient was pulmonary sepsis or hemoptysis the dominant factor responsible for death. Because so little benefit would accrue the patient from extended pulmonary resection in the presence of a positive biopsy, it would seem to be a definite contra-indication to pulmonary resection for lung carcinoma.

#### RESUMEN

La biopsia de los ganglios escalénicos tiene un valor demostrado en el diagnóstico de las afecciones malignas pulmonares y asimismo, tiene significación pronóstica.

El término medio de sobrevivencia que es de esperarse en 24 casos después de biopsia positiva, es de 3.6 meses con una sobrevivencia de la mayoría de tres o menos meses.

Las complicaciones de la enfermedad ya se encontraban presentes cuando se hizo el diagnóstico. Las complicaciones pulmonares son a menudo opacadas por las metástasis extrapulmonares.

En ningún enfermo fué la infección o la hemoptisis el factor dominante responsable de la muerte. Puesto que poco beneficio puede esperarse para el enfermo a quien se haga una resección pulmonar extensa cuando hay una biopsia de ganglios escalénicos positiva, debe considerarse ésta como una contra-indicación definida para la resección pulmonar por carcinoma.

#### RESUME

La biopsie des ganglions préscaléniques est de valeur démontrée dans le diagnostic de cancer pulmonaire et a vraisemblablement une signification pronostique. La survie moyenne dans 24 cas après biopsie positive est de 3.6 mois avec une majorité que ne survit que trois mois ou moins. Des complications de l'affection existaient souvent déjà au moment du diagnostic. Des complications pulmonaires furent souvent masquées par des métastases extra-pulmonaires. Chez aucun malade la gangrène pulmonaire ou l'hémoptysie ne furent les facteurs dominants responsables de la mort. Le malade semble tirer si peu de bénéfice d'une résection pulmonaire étendue quand la biopsie préscalénique est positive qu'elle devrait être considérée comme une contre-indication formelle à la résection pulmonaire dans le cancer pulmonaire.

## ZUSAMMENFASSUNG

Biopsie des Skalenus-Lymphknotens ist nachgewiesenermaßen von Wert bei der Diagnose pulmonaler bösartiger Veränderungen, und hat ebenfalls prognostische Bedeutung. Die mittlere Lebenserwartung bei 24 Fällen nach positiver Biopsie beträgt 3.6 Monate, wobei die Mehrheit 3 Monate oder weniger lang lebte. Die Komplikation der Erkrankung lag oft vor dem Zeitpunkt der Diagnose.

Die pulmonale Komplikationen wurden oft überschattet durch extrapulmonale Metastasen. Bei keinem Kranken war eine von der Lunge ausgehende Sepsis oder Haemoptyse der dominierende, für den Tod, verantwortliche Faktor. Weil dem Kranken so wenig Nutzen erwachsen würde von einer ausgedehnten Lungenresektion bei Vorliegen einer positiven Skalenus-Biopsie, dürfte sie eine eindeutige Gegenindikation darstellen für die Lungenresektion von Carzinomen.

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## Studies in Emphysema

### II. Bedside Versus Laboratory Estimations of Timed and Total Vital Capacity and Diaphragmatic Height and Movement

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As part of a longitudinal study of domiciliary members with emphysema, we have had an opportunity to compare estimations of timed and total vital capacity, using the McKesson-Scott Vital Capacity Apparatus, and diaphragmatic height and movement elicited by physical examination with those measurements obtained in the pulmonary function and x-ray laboratories.

The patients used in this study had typical histories of emphysema without bronchial asthma; most had associated bronchitis, but three had no cough preceding the onset of dyspnea on exertion and continue to be free of cough and expectorate no sputum.

The clinical examiner recorded chest wall movement, spine movement, use of accessory respiratory muscles, height and motility of the diaphragm, presence, absence or degree of reversible bronchial obstruction and intercostal hypertrophy, both before and after nebulization with bronchodilators administered by intermittent positive pressure for 10 minutes. The clinical estimation of timed and total vital capacity was performed with the original McKesson-Scott Vital Capacity Apparatus. A fine wire was soldered to the pointer on the dial, and the rulings were carried to the outside cover to facilitate reading of timed vital capacity as the pointer moved.

We have found that timed vital capacity readings in which the time is estimated by the examiner do not vary appreciably from those obtained using the electrically or mechanically timed machines. A trained observer can estimate 30 seconds plus or minus one second.

As soon as the examiner had concluded the pre-nebulization studies, each patient was tested on the Gaensler Timed Vitalometer and on the 13-liter Collins Respirometer by an experienced technician. The nebulization was then performed and both the clinical and laboratory studies were repeated. At this time roentgenograms of the chest were made during full inspiration and expiration so that diaphragmatic movement could be measured.

Early in our study we were disturbed by the marked variation in the same patients both before and after nebulization when estimations of total vital capacity using the McKesson-Scott, the Gaensler Timed Vitalometer and the Collins Respirometer were compared. It soon became apparent that the variation between the results obtained on the more elaborate laboratory devices was as great as those obtained on the portable McKesson-Scott apparatus (Table 1).

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TABLE 1—COMPARISON OF VARIATION IN TOTAL VITAL CAPACITY ON THREE MACHINES

Total V.C.	No.	Min.	Max.	Mean	$\sigma$
1. G. vs. C.	31	0	600	216	171.8
2. McK. vs. C.	33	20	590	247	173
3. McK. vs. G.	32	0	600	197	179
G.—Gaensler	C.—Collins	McK.—McKesson-Scott			

Accordingly, 21 patients were tested for total vital capacity on the Gaensler Timed Vitalometer. Each blew at least three pairs of observations with a rest period of 30 to 60 minutes between. All testing was done by the same trained technician, who attempted to get maximal effort from each patient at each test. The results demonstrated variations in the same patients of from 0 to 500 cc. There was no correlation between these variations and the successive measurement.

TABLE 2—COMPARISON IN VARIATION IN VITAL CAPACITY RESULTS, PAIRED OBSERVATIONS ON THE SAME PATIENTS

Total V.C.	No.	Min.	Max.	Mean	$\sigma$
Gaensler (only)	64	0	500	180	136

In order to be certain of maximal effort on the part of the patient, one who showed variation of 500 cc. between successive pairs of total vital capacity estimations was used in two hypnotic experiments. At the first session he was placed in moderately deep hypnosis and was given post-hypnotic suggestions, that he would be able to make a maximal effort during the test. Since he did no better following these suggestions than he had done without them, a week later he was placed in deep hypnosis and the suggestions were given and the test performed while in the hypnotic state. Again there were similar wide variations between pairs of tests.

We have compared the measured diaphragmatic movement on x-ray films with the recorded clinical estimation 130 times, and variations of 1 cm. or less were obtained in 41.5 per cent. The mean difference was 1.34 cm., with a range of from 0 to 4.5 cm., with 93.8 per cent falling within 0 to 3 cm.

In order to check on the significance of these differences, we obtained films during two separate inspirations and expirations on a small series of patients. Variations in the measurement of diaphragmatic movement between the two series of films was as great in several of the patients as those reported above. Several factors may be involved.

The differences may reflect changes such as those reported above for vital capacity, since differences of as much as 20 per cent between successive tests must be associated with changes in diaphragmatic movement. In addition, four different technicians in the x-ray department made the exposures and may not have been able to get the patient to make a maximal effort.

#### SUMMARY

These studies demonstrate that for clinical purposes bedside tests for timed and total vital capacity are as valuable as the more time-consuming pulmonary function laboratory tests. The bedside method has the additional advantage that the clinician

in charge of the patient observes the test, can watch the movement of the dial and can determine the degree of cooperation of the patient.

The wide variations between paired tests in patients with emphysema make judgment of the effects of therapy difficult. These patients respond subjectively to attention and frequently report improvement which cannot be demonstrated objectively. Objective evidence of improvement requires differences of at least 20 per cent in timed or total vital capacity. One cannot use the standard error figures for normals for the evaluation of therapy in patients with emphysema.

Our studies additionally demonstrate that the height and movement of the diaphragms can be measured at the bedside with sufficient accuracy for clinical purposes and that recourse to elaborate x-ray examinations yields no more significant information.

#### RESUMEN

Estos estudios demuestran que para los fines clínicos, las pruebas a la cabecera del enfermo para capacidad vital total y por segundos, son tan valiosas como las pruebas de función de laboratorio que son mucho mas prolongadas. El método a la cabecera tiene la ventaja agregada de que el médico a cargo observa la prueba, puede observar el movimiento de la aguja y determinar el grado de cooperación del enfermo.

Las amplias variaciones entre pruebas pareadas de enfermos con enfisema hace que el juicio sea difícil. Estos enfermos responden subjetivamente a la atención y frecuentemente relatan mejoría que no se comprueba objetivamente. La evidencia objetiva de la mejoría requiere diferencias de por lo menos 20 por ciento en la capacidad vital total y en la capacidad por segundos.

No se pueden usar las cifras estandard de error para los normales en los enfermos de enfisema.

Nuestros estudios adicionales demuestran que la altura y el movimiento del diafragma puede medirse a la cabecera con suficiente exactitud para fines clínicos y el recurrir a complicados exámenes a los rayos X, no dan mayor información de importancia.

#### RESUMÉ

Ces études démontrent que pour les besoins de la clinique, les tests de capacité vitale (volume expiratoire maximal seconde et capacité totale) sont aussi valables au lit du malade qu'au laboratoire où ils prennent bien plus de temps. La méthode pratiquée au lit du malade a l'avantage supplémentaire que c'est le médecin qui s'occupe du malade, qui observe le test, peut faire attention au mouvement du cadran et peut déterminer le degré de collaboration du malade.

Les larges variations entre tests jumelés chez des malades atteints d'emphysème rendent difficiles le jugement des effets thérapeutiques. Ces malades répondent subjectivement à l'attention qu'on leur porte et font fréquemment état d'une amélioration qui ne peut être démontrée objectivement. Une preuve objective de l'amélioration demande des différences d'au moins 20% dans la capacité vitale totale et maximale seconde. On ne peut pas utiliser les taux d'erreur habituellement considérés comme normaux pour l'estimation de la thérapeutique chez les malades emphysemateux.

Ces études montrent en outre que la hauteur et le mouvement du diaphragme peuvent être mesurées au lit du malade avec une précision suffisante pour des desseins cliniques et que le recours à des examens radiologiques approfondis n'apporte pas de renseignement plus significatif.

#### ZUSAMMENFASSUNG

Diese Untersuchungen demonstrieren, daß für klinische Zwecke Bestimmungen der zeitbegrenzten wie der totalen Vital-kapazität an im Bett liegenden Kranken den gleichen Wert haben, wie die mehr zeitraubenden Lungenfunktionsprüfungen im Laboratorium. Die Untersuchungen bei Bettpatienten haben den zusätzlichen Vorteil, daß der Stationsarzt des Patienten den Test durchführt, die Zeigerbewegungen beobachtet und bestimmt, in welchem Maß der Patient sich anstrengen darf.

Die erheblichen Schwankungen bei wiederholten Testen bei Kranken mit Emphysem gestalten die Beurteilung der Wirkungen der Therapie schwierig. Die Patienten reagieren subjektiv auf die ihnen geschenkte Beachtung und geben häufig eine Besserung an, die objektiv nicht nachzuweisen ist. Der objektive Nachweis einer Besserung erfordert Unterschiede von wenigstens 20% in der zeitbegrenzten oder der totalen Vitalkapazität. Man kann die für normale Versuchspersonen gültigen Standard-schwankungen nicht anwenden bei der Beurteilung der Therapie bei Kranken mit Emphysem.

Unsere Untersuchungen demonstrieren darüber hinaus, daß die Höhe und die Verschieblichkeit der Zwerchfelle am Krankenbett mit genügender Genauigkeit für klinische Zwecke gemessen werden kann, und daß der Weg der Erhebung röntgenologischer Untersuchungen keine beträchtlich besseren Informationen ergibt.

## Triamcinolone in Asthma and Secondary Bronchospastic Pulmonary Emphysema

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Triamcinolone possesses, in common with other clinically useful corticosteroids, the striking ability to curtail the allergic reaction irrespective of wide differences in precipitating factors and in responsiveness to other forms of therapy. Palliative effects in bronchial asthma have been induced promptly following the administration of comparatively small doses<sup>1-4</sup> and it is a recent observation that with a conservative approach to dosage, satisfactory relief of respiratory distress may be obtained with few, if any, serious complications.<sup>4,5</sup> The extensive systemic actions of all corticosteroids, which give rise to unwanted, as well as to therapeutic effects, dictates that hormonal therapy should not be employed until all customary remissive measures have proved inadequate.<sup>6</sup> The precaution of frequent clinical observation during treatment to determine symptomatic response, the possible presence of adverse reactions and the need for adjustment of dosage applies to triamcinolone as it does to all corticosteroid compounds developed thus far. Because the decision to use a corticosteroid in bronchial asthma depends upon the severity of the respiratory embarrassment compared with the potential risk of hypercorticism, its administration is usually confined to the chronic intractable case. It is refractory asthma, complicated by bronchospasm and emphysema, with which this investigation is particularly concerned.

The impressive subsidence of clinical symptoms of asthma achieved with triamcinolone implies a somewhat parallel improvement in respiratory function, objectively demonstrated as a change in vital capacity and in expiratory flow rate. Measurements taken before, during, and after corticosteroid medication provide objective evidence of the reversal of respiratory difficulty attributable to drug action, thereby disclosing the extent of permanent pathological changes in lung parenchyma. Because many patients show a normal vital capacity but require considerably longer than the normal 3 seconds to expire this volume, the total expiration time is a useful index of pulmonary function. Together determinations of vital capacity and total expiration time offer information for a fuller knowledge of functional ability to ventilate properly. The use of these objective criteria in conjunction with subjective experience of ameliorative action constitutes a sound basis for the total assessment of corticosteroid benefits.

Changes in vital capacity and in total expiration time induced with triamcinolone were observed in a limited group of patients with serious ventilatory insufficiency due to long-standing bronchial asthma with chronic bronchitis and progressive pulmonary emphysema complicating

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The triamcinolone employed in this study was supplied as Kenacort through the courtesy of Dr. E. C. Reifenstein, Jr., E. R. Squibb & Sons, New York, New York.

the treatment problem. Clinical features were typical although of varying intensity leading to varying degrees of dysfunction and disability. Subjective signs of improved pulmonary function were anticipated in view of the well-established suppressive influence of corticosteroids on respiratory symptoms due to the exudative processes of an allergic reaction. There was a question, however, of the satisfactory maintenance of respiratory comfort over a reasonable period without the use of bronchodilators or aminophylline in cases with a significant degree of irreversible disease and where an appreciable dependence upon these medications had developed over a period of years. Since the hyperphysiologic effects of corticosteroids have become an important consideration, an attempt was made to determine minimal therapeutic and maintenance dosage levels and the appearance and nature of unwanted effects with otherwise satisfactory palliative dosage. Measurement of the functional defect prior to treatment and at regular intervals during the administration of triamcinolone served as a more reliable guide to the adequacy of the management program and to the effectiveness of therapy than subjective estimates of symptomatic relief. This in itself is important to the physician since it permits close supervision of the patient, materially reducing the hazard of serious adverse reactions with the potent corticosteroids in current usage.

#### *Clinical Material*

The cases presented were encountered in private practice. Most patients were treated for dyspnea and wheezing, the common features of acute respiratory distress. Nine ranged in age from 51 to 75 years and the remaining two were 28 and 35 years of age. Bronchial asthma with typical chest findings was present in all cases but one, with the fundamental pulmonary disease complicated by arteriosclerotic heart disease in three instances and by secondary emphysema, cor pulmonale, chronic bronchitis, obesity, diabetes and hypertension, singly or in combination, among the older patients. Treatment of the recurrent acute attack was previously carried out in conservative fashion with rest, bronchodilators, aminophylline, and iodides.

Five patients had had thorough allergy surveys and received desensitization therapy with some improvement, which, however, was not satisfactorily maintained. Paroxysmal asthmatic attacks would erupt over a sustained plateau of chronic asthma. The remaining patients had been studied sporadically over many years, but had no complete survey at the time this study was begun. Because of the severity of their symptoms, therapy with triamcinolone was instituted and the allergy survey was delayed until the patient's condition improved on the non-specific therapy, which was being evaluated. It should be emphasized that in all instances of asthma, a comprehensive allergy investigation is basic to the regular management of the patient.

Triamcinolone was given because prior measures proved inadequate or when the patient was severely ill.

Therapy with the triamcinolone was initiated with doses of 4 milligrams four times daily. No patient required larger initial doses and benefits were almost invariably apparently in responsive cases within 24 hours

of the onset of treatment. The schedule best suited to most individuals was 4 mg. q.i.d. for one to two weeks followed by daily doses of 4, 2, 4, and 2 mg. for one week, then by 2 mg. q.i.d. for one week and by alternate doses of 2, 1, 2, and 1 mg. for still another week. Further reductions in dosage were instituted with 1 mg. q.i.d. given for one week and 1 mg. b.i.d. for one week until finally a low of 1 mg. daily was achieved. The lowest maintenance dose satisfactory from both objective and subjective viewpoints was 1 mg. daily following a six to eight week period of more intensive therapy. All patients were seen weekly until maintenance dosage was established, the drug discontinued or therapeutic failures re-evaluated for other than allergic etiology.

Although the treatment period covered by this report was comparatively short, careful consideration was given to the appearance of signs of hypercorticism and other unwanted effects associated with the administration of the various corticosteroid compounds. Sulkowitch tests were regularly performed to disclose early effects on bone. No attempt was made, however, to restrict salt intake or to administer potassium supplements while triamcinolone was prescribed in the aforementioned dosage.

### Results of Treatment

Improvement in pulmonary ventilation and a good to excellent symptomatic response to triamcinolone was observed in all but one patient treated during this investigation (Table 1). Some patients experienced sufficient clearing of the airway and amelioration of distress to discontinue medication. None required continuous therapy with bronchodilators while taking the corticosteroid. Benefits of treatment included a notable absence of acute paroxysmal attacks, an appreciable increase in alertness, and an ability to remain active and to continue performance of duties necessary for employment or care of the household.

Improvement in vital capacity, ranging from 100 to 700 cubic centimeters, and lessened expenditure of respiratory energy eased the respiratory work load. Total expiration time in this series was reduced from one to five seconds during treatment. Subjective reports of more comfortable respiration, a sense of well being, and increased tolerance for

TABLE 1—IMPROVEMENT IN VENTILATION AND SYMPTOMATIC RESPONSE TO TRIAMCINOLONE IN BRONCHIAL ASTHMA AND EMPHYSEMA

Patient	Sex	Period of Treatment	Vital Capacity (cc.)		Total Expiration 3-Second V.C. Time (sec.) (% of total V.C.)				Control of Symptoms
			Initial	Final	Initial	Final	Initial	Final	
S.B.	F	8 weeks	2750	2860	12	7.5	66	77	Excellent
D.G.	M	8 weeks	2350	2450	5	3	84	100	Good
M.B.	F	8 weeks	1700	2400	8	4	68	95	Excellent
N.B.	M	3 weeks	2600	3100	16	14	53	64	Good
G.G.	M	6 weeks	3050	3150	4	3	94	100	Excellent
H.B.	M	8 weeks	3000	3300	5	4	84	98	Excellent
I.F.	M	7 weeks	3150	3600	9	6	82	85	Excellent
W.K.	M	4 weeks	3350	3350	15	10	70	86	Good
D.F.	F	7 weeks	3300	3550	12	7	87	90	Good
M.H.	F	4 weeks	2900	3000	6	5	81	88	Excellent
J.S.	M	5 weeks	1600	1800	2	3	100	99.9	Poor*

\*Patient in heart failure, no worsening of symptoms while taking triamcinolone.

physical activity frequently exceeded that which might be anticipated from objective findings. This, however, was considered a reflection of the prominence of subjective influences in bronchial asthma and in other allergic states as well.

The single patient showing a poor response to triamcinolone was one with hypertensive and arteriosclerotic heart disease, and left bundle branch block, who experienced recurrent episodes of left ventricular failure. These attacks were associated with a bronchospastic element, as indicated by prolonged expiratory wheezing. This patient was maintained in partial compensation with digitalis, diuretics, and aminophylline. It is noteworthy that while the administration of the corticosteroid failed to ease the respiratory difficulty, (which had no allergic basis, but was due to left ventricular insufficiency), the patient's status was in no way affected by the triamcinolone.

An important observation during this study was the uniformity and promptness of response to triamcinolone where respiratory disease was referable to an allergic etiology. In these cases, some improvement was almost invariably apparent within 24 hours of institution of therapy with marked benefit achieved within the first 48 hours. The absence of detectable change within this period was considered sufficient indication for re-evaluation of precipitating factors and original diagnosis. In this way, the rapid palliative action of triamcinolone lends itself remarkably well to the differential diagnosis of bronchial asthma and an illustrative case history is presented below.

Complications of treatment were few and not of serious consequence. Loss of weight occurred occasionally during the first two weeks, but this was subsequently regained. Gastrointestinal discomfort was a complaint in two cases disappearing with the concomitant use of antacids. On the prescribed dosage schedule, none of the patients in this series developed edema, peptic ulcer, elevated blood pressure, glycosuria or mental depression. One diabetic, however, previously controlled by diet alone required oral 1-phenethylbiguanide for control of glycosuria while taking triamcinolone. All Sulkowitch tests remained within normal limits and there was no evidence of electrolyte disturbance with short-term therapy. No Cushingoid changes were observed other than hirsutism in one individual during the seventh week of treatment.

### *Case Reports*

*Case 1:* L. M., a male, 54 years of age, was referred with a complaint of constant chest pains of several months duration. For a period of four months, he had been treated for bronchial asthma by desensitization injections and other conventional medications, with no improvement. There was no dyspnea on exertion and no palpitation, but he was troubled by an inconstant cough during this time. Occasional wheezing was reported. On several occasions, he was instructed to take nitroglycerin, reacting with violent headache, but without improvement in chest discomfort or wheeze. X-ray examination of chest, esophagus and gastrointestinal tract was negative. He had been treated with chlorothiazide and reserpine without benefit. Past history included elevated blood pressure of ten year's standing and nasal polypectomy five years earlier.

Physical examination of this 217 pound male revealed blood pressure of 190/94, grade I fundal sclerosis, but no abnormalities of heart or lungs. The heart showed no enlargement on x-ray and the lungs appeared normal. Electrocardiogram demonstrated low T-waves. Blood count was normal and only one eosinophile was encountered in the differential white cell study. A nasal smear revealed two eosinophiles. A decholin circulation time, arm to tongue, was 16 seconds.

At this time, symptoms failed to respond to aminophylline, ephedrine, bronchodilators, reserpine, antispasmodics, tranquilizers or diuretics. Spirometric study revealed a vital capacity of 3630 cc. with a timed respiration of 66 per cent in one second, 86 per cent in two seconds, and 91 per cent in three seconds. Triamcinolone was then given in doses of 4 mg. four times daily. After one week of treatment, no improvement was detected. Since all previous cases of bronchial asthma responded to triamcinolone within a week, this case was considered unusual and worthy of re-evaluation. A second questioning of the patient elicited the information that wheezing generally occurred while he reclined on his right side. He was then re-examined in a sitting position and no wheeze was heard. Some 15 minutes after lying on his right side, however, the patient began to wheeze with prolonged expiratory phase, and sibilant rales were heard over the right base as well as the trachea. Over the left chest a distant wheeze was detected, probably transmitted. When the patient sat up, the wheeze disappeared within two minutes. From these findings and the failure to respond to triamcinolone, the diagnosis suspected was a bronchial polyp. The patient was admitted to the hospital and a bronchoscopy was performed which disclosed a benign polypoid growth in the right lower bronchus.

*Comment:* Since triamcinolone had so consistently relieved asthmatic symptomatology, a therapeutic failure was considered a feature for differential diagnosis and indicated a need for further evaluation of physical signs. The uniformity and promptness with which triamcinolone elicits a response in bronchial asthma of allergic origin lends itself to diagnosis as well as therapeutic applications.

*Case 2:* H. B., a male, 66 years of age, is a case in point demonstrating the pronounced benefits which may be achieved with triamcinolone. This patient revealed a history of bronchial asthma extending back as far as 25 years and presently complicated by chronic bronchitis and emphysema. During the past three years, he had experienced increasing difficulty in breathing and in the month prior to this examination, respiratory distress had worsened considerably. Although cough was severe, hemoptysis was not observed. Physical examination disclosed an emphysematous chest with numerous, well-defined rhonchi and coarse rales. The nasal mucosa was pallid. X-ray findings included emphysema and bronchial fibrosis, chiefly at the bases. Electrocardiogram demonstrated pulmonary P waves ( $P_{2,3}$ ). Spirometric evaluation showed a vital capacity of 3000 cc. with a timed respiration of 47 per cent in one second, 78 per cent in two seconds, and 84 per cent in three seconds. Pre-treatment expiratory time was 5 seconds.

Triamcinolone was prescribed in doses of 4 mg. q.i.d. for the first two weeks, followed by alternate doses of 4 mg., 2 mg., 4 mg., and 2 mg. daily for 2 weeks, then 2 mg. q.i.d. for 2 weeks and 4 mg. b.i.d. for 2 weeks. Spirometric studies at the end of the 8-week treatment period demonstrated a vital capacity of 3300 cc. and a timed respiration of 50 per cent in one second, 78 per cent in two seconds, and 98 per cent in three seconds. Expiration time was reduced to 4 seconds. Subjective improvement included relief of dyspnea and acute respiratory difficulty, easing of cough, lessening of fatigue with exertion, and a more optimistic mental state. No unwanted effects were observed in this patient during treatment. A gain in weight of 2 lbs. in 8 weeks was recorded. Blood potassium, sodium and chloride levels remained unchanged and no edema or gastric upset was experienced.

*Comment:* Response to treatment was considered excellent in view of the long-standing disease and lung pathology.

### Discussion

Spirometric studies in bronchial asthma, a fundamental guide to the severity of the acute attack and to the degree of irreversible pulmonary insufficiency, may be profitably applied to the determination of the adequacy of corticosteroid dosage, individual responsiveness and competency of the treatment program. Subjective expression of symptomatic relief is almost universally expected in asthma attributable to allergic factors but because of the prominent subjective and emotional elements in the disease process itself, this estimate of drug benefits leaves much to be desired. Changes in vital capacity and expiration time brought about by treatment with corticosteroids provide a more substantial measure of improvement in functional ability to ventilate properly and regular determinations of lung volumes and expiratory flow rate enable the close patient supervision required by potent medication. While a variety of ventilatory function tests may be employed for similar purposes, vital capacity together with total expiration time offer useful information and are measured easily and without great expense with any simple spirometer.

Improvement in vital capacity of from 100 to 700 cubic centimeters was achieved in a limited group of patients with intractable asthma, bronchospasm and progressive emphysema treated with triamcinolone. Expiration time was reduced from one to five seconds. Antibiotics were administered where infection was manifested, but aside from this, many patients could be maintained in respiratory comfort without additional medication although all had been dependent upon bronchodilators and aminophylline prior to corticosteroid therapy. In most instances, recurrent acute situations were not observed during this study. Certain findings are particularly worthy of com-

ment. The palliative action of triamcinolone where bronchial asthma is attributable to allergic sensitivity is almost invariably apparent within 24 hours reaching a maximum effect usually within 48 hours of initial dosage. Failure of response is a firm indication for re-examination of the patient and re-appraisal of physical findings. Once the respiratory distress is controlled minimal maintenance dosage should be established and individualized. While triamcinolone is a potent and useful corticosteroid, in the total management of chronic bronchitis and emphysema, the combination of aminophylline, ephedrine and iodides is frequently valuable adjunctive therapy. Like all corticosteroids, triamcinolone should not be employed for the acute paroxysms of asthma since the time-lag in suppressive action is too great for the immediate clearing of the airway that is desired. Acute therapy must be carried out as usual with aminophylline, epinephrine, isopropylarterenol and other similar and conventional medications. As early as feasible, allergy surveys and the use of elimination and desensitization procedures should be carried out in all instances of allergic states.

#### SUMMARY

A limited group of ten patients with intractable asthma, bronchospasm and pulmonary emphysema were observed during treatment with triamcinolone. Changes in vital capacity and total expiration time, measured at regular intervals, provided objective evidence of improvement in pulmonary function attributable to drug action. Subjective expression of benefits tended to surpass objective findings and included elimination of acute paroxysmal attacks, an increase in alertness and an ability to actively perform duties required by employment or care of the household. Complications of treatment observed over a period of two months were few and of no serious or lasting consequence. None of the patients in this series developed edema, peptic ulcer, elevated blood pressure, glycosuria or mental depression. The usefulness of simple spirometric studies as an index of adequacy of corticosteroid dosage, individual responsiveness and competency of treatment program is suggested. The application of the prompt and consistent palliative action of triamcinolone to the differential diagnosis of bronchial asthma is discussed and two case histories are presented.

**ACKNOWLEDGEMENT:** The author extends his thanks to Martin G. Waldman for his valuable assistance in the spirometric studies and calculations.

#### RESUMEN

Un grupo reducido a diez enfermos de asma intratable con broncoespasmo y enfisema pulmonar, se observaron durante tratamiento con triamcinolona. Los cambios de la capacidad vital, y expiración total, medidos a intervalos regulares, proporcionaron muestra objetiva de mejoría atribuible a la droga. La mejoría subjetiva fué mayor que la objetiva comprendiendo la eliminación de los ataques agudos de los paroxismos, aumento de actitud alerta, y capacidad para realizar el trabajo de sus empleos o del hogar. Las complicaciones observadas durante un período de dos meses fueron pocas y no serias o duraderas. Ninguno de los enfermos de esta serie tuvo edema, úlcera péptica, hipertensión, glucosuria, o depresión mental.

La utilidad de los estudios espirométricos simples como un índice de la dosificación adecuada, la respuesta individual y la competencia del tratamiento se sugiere. La aplicación de la acción inmediata y sólidamente paliativa de la triamcinolona para el diagnóstico diferencial del asma, es motivo de discusión en un caso que se presenta.

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## Parenchymatous (Bronchopulmonary) Manifestations of Pulmonary Bilharziasis\*

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In a previous communication, one of us (Sami),<sup>1</sup> reviewed the pulmonary manifestations of schistosomiasis.\*\* While the vascular ones are now fairly well-defined, the "parenchymatous" or "bronchopulmonary," are far from being definite. The necessary correlation between pathological findings and clinical features, and satisfactory criteria for diagnosis, have yet to be established. The present paper is an attempt at giving this elusive aspect a more factual and concrete basis.

Madden<sup>2</sup> in 1907, stated that ova had been discovered in the fine capillaries of the walls of the alveoli, even giving rise to a form of chronic interstitial pneumonia.

Sorour<sup>3</sup> in 1928, described bilharzial tubercles in the peribronchial and perivascular connective tissue of the lungs, followed by "lamellar whorly fibrosis." He described an "endobronchitis obliterans," as well as a diffuse proliferation of the alveolar epithelium which later underwent desquamation, all resulting in a fleshy pneumonic lobule.

Azmy and Effat<sup>4</sup> described two cases of bilharziasis in 1932 in which there was dilatation of the pulmonary artery, as well as chronic bronchitis and emphysema. These were the first cases described clinically of pulmonary bilharziasis.

Mainzer<sup>5</sup> reported the following forms of pulmonary bilharziasis: (1) an acute initial phase; (2) a chronic type resulting in chronic bronchitis, bronchiectasis, emphysema and fibrosis; (3) a latent radiological form characterized by miliary mottlings or a "birch-broom" appearance; (4) *cor pulmonale*, and (5) asthma.

In 1937, Day<sup>6</sup> described a pulmonary form of bilharziasis characterized by cough, sputum, crepitations and small rounded shadows in the x-ray film, associated with hepatic bilharziasis, and a cardiopulmonary form in which in addition there was enlargement of the pulmonary artery.

In 1938, Shaw and Ghareeb<sup>7</sup> published an exhaustive pathological study. They found ova in the lungs in one third of 382 cases of schistosomiasis. Parenchymatous bilharzial tubercles were present in only 86 per cent of the cases. The ova were scanty, and the tubercles invisible to the naked eye. These tubercles were due to the escape of embolised ova from the arterioles into the bronchiolar wall or into the alveoli on either side. They were 250 to 375 microns in diameter. Outside them, they stated, "there was no tissue reaction, and there were normal alveoli in immediate contiguity with the tubercle." With heavier infestation with ova, there were vascular lesions in addition to the parenchymatous ones, but in none of the cases was there gross parenchymatous changes apart from those described above. A totally different type of lesion, which was, however, much less frequent, was that produced by worms. When em-

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\*\*Synonym: bilharziasis.

bolised worms died in the lung, they caused a characteristic necrotic and focal pneumonia which appeared as an opaque white round or elliptical area of consolidation, 0.1 to 0.5 cm. in diameter. The walls of the surrounding alveoli were congested and the lumen filled with eosinophils, some polymorphs and histiocytes and a few red cells. Later, organisation occurred, the dead worm was calcified and enclosed in a capsule of scar tissue.

Warraki<sup>4</sup> in 1950, in a pathological study of bronchitis, met with two cases in which the lungs were heavily infested with bilharzia. Numerous tubercles containing ova were present and there was fibrous thickening of the arteriolar walls. In both of them, serial sections of the bronchi did not show bilharzial lesions.

Nour-el-Din and Baz<sup>5</sup> in 1954, studied 62 cases of bilharzia, 48 of which were simple cases of urinary bilharzia and 14 had hepatosplenic involvement. Ova were found in the sputum in 22 cases, of which four complained of haemoptysis. Ayerza's syndrome was present in four, some rhonchi and crepitations were present in 12, and five had asthma. X-ray films showed mitralisation or cor pulmonale in several, with a nodular appearance of the pulmonary arteries.

Erfan, Moussa et al<sup>6</sup> in 1957, gave a detailed account of "bronchopulmonary bilharziasis," and described: (1) alveolar and pleural forms, including pneumonitis, fibrosis and emphysema; (2) bronchial forms, including asthma, asthmatic bronchitis and bronchiectasis, and (3) milary, nodular and granulomatous forms.

No attempt has been made here to review the numerous articles, mostly by Egyptian authors, which deal with the cardiovascular type of pulmonary bilharziasis. In most of these, parenchymatous pulmonary clinical manifestations are conspicuous by their absence. In a few, such changes are described.

The present work consists of two parts. In the first, 57 cases of bilharzia—all of which had urinary infestation with intestinal infestation in three—had their lungs examined clinically, radiologically and bronchographically. Forty-six were admitted because of symptoms referred to the lungs, and 11 had no such symptoms.

The second part consisted of a mass radiography investigation of 6375 of the inhabitants of a rural area near Cairo (Qualioub). As part of the survey, which was conducted in conjunction with the Qualioub Demonstration and Training Centre, every third case was examined for parasites, including bilharzia. An analysis of the pulmonary findings in the positive cases was then made.

### *Part I*

*Clinical Features.* Forty-six of the total 57 men had been admitted because of chest symptoms, and 11 had none. Of the 46 with chest symptoms, 14 had symptoms and/or signs of bronchitis, 26 had asthma or "asthmatic bronchitis," five had generalized emphysema, and one was admitted because of haemoptysis.

Of the cases of "bronchitis," two had localized crepitations, the rest had no sign or had generalized rhonchi.

All the cases were put under antibilharzial treatment with tartar emetic and eight showed improvement or disappearance of the symptoms or signs during their hospital stay. The rest (38 cases) showed no change.

*Radiological Features.* Of the 11 asymptomatic cases, two had parenchymatous shadows. In one of the latter, the shadow consisted of a peripheral small calcification, and the tuberculin test was positive. In the other, the shadow consisted of a light opacity in the middle zone. The tuberculin test in this case was not reported.

Of the 46 symptomatic cases, x-ray films were normal in 13, "increased markings" were reported in 27 and six had parenchymatous opacities. These were localized, fluffy or strandy and of minimal extent.

*Bronchography* was performed in 18 cases of whom 14 had normal findings, and four showed minimal bronchiectatic changes and involved only one or more bronchi in a subsegmental distribution. Two of the four cases had as well generalized "asthmatic bronchitis." In one case, the asthma was related to the ingestion of eggs.

*Ova in the sputum.* The 24-hour sputum is mixed with an equal volume of 4 per cent sodium hydroxide, and kept for two hours in a warm water bath, until the mucus is dissolved. Specimens from the sediment were examined directly under the microscope for the bilharzia ova. Twenty-nine of the cases had the examination done and the result recorded. Three gave positive results.

*Effect of antibilharzial treatment.* After admission and initial investigation all patients received a course of antibilharzial treatment consisting of 12 intravenous injections of 6 per cent tartar emetic, given every other day, in doses of one-half to 2 cc. and the effect on the clinical picture and on the plain x-ray film noted.

Clinically the effect of treatment was recorded in 22 cases of whom 16 had asthma or asthmatic bronchitis, and six had simple bronchitis with localized or generalized signs. Disappearance of symptoms and signs by the end of treatment was noted in four, all of whom are among the simple bronchitic group. Improvement was noted in 10 (eight asthmatics and two bronchitic), but in three this was transient. In six there was no change (five asthmatics, one bronchitic). Two (both asthmatic), were exacerbated during or after treatment. The significance of these findings will be discussed later.

## Part II

Of 6375 individuals who were x-rayed, 363 (5.6 per cent) showed radiological pulmonary changes.

Of the 2783 individuals who had parasitic examination 198 (11.1 per cent) had bilharzia. Of these 198 cases, eight (4.4 per cent) showed pulmonary radiological pathology. This is less than the figure for the general population of the district as shown above, but the difference is not significant. In this study, therefore, bilharzial infection was not associated with increased incidence of abnormal pulmonary shadows.

## Discussion and Conclusions

From the review of the observations of previous workers, as well as the present series of cases, it is seen that pulmonary manifestations in cases of bilharzia are numerous and variable. It is natural, but by no means necessarily true, to assume that any abnormality present in the lungs in a case of bilharzia is causally related. as a

matter of fact, such were the conclusions in several of the reviewed papers. If we keep in mind, however, that on the one hand bilharzial affection is very widespread in the countries from which these reports come, and on the other hand that most of these observations were made on hospital patients who naturally only present themselves because they seek treatment from some troublesome condition, we cannot escape the strong suspicion that many of these manifestations may be those of associated conditions, having no causal relation to the bilharzia itself.

The one diagnostic criterion for pulmonary bilharziasis during life is the detection of bilharzia ova in the sputum. Even this, however, does not necessarily mean that all the pulmonary findings in the case are bilharzial. Another condition which has to be present is that these findings should be compatible with the pathology of bilharzial lesions, particularly as shown in the most complete study available to us, that of Shaw and Ghareeb.

In addition to this, we have to recognize the possible allergenic influence of bilharzial worms and ova, and try carefully to assess its role.

Accordingly, the pulmonary manifestations in bilharzia may be divided as follows:

**A. Vascular Component:**

*Various grades of cor pulmonale. Possible radiological arterial changes: nodulation, tortuosity.*

**B. Parenchymatous component:**

1. Ova in sputum: (a) without other manifestations, (b) with manifestations compatible with bilharzial pathology, (c) with associated manifestations (v.i.).

2. No ova in sputum: (a) manifestations compatible with bilharzial pathology, (b) associated manifestations.

3. Allergic manifestations: *Asthma, Loeffler's syndrome.*

The vascular manifestations are now well defined, and are not the subject of the present study. It is essential to stress, however, that the essential lesion of pulmonary bilharzia originates in the arterioles of the pulmonary circuit, in which or in their immediate vicinity it lies. This cardinal fact makes it highly improbable that diffuse fibrosis, would take place, as one would expect that long before that the widespread arteriolar affection would lead to predominant vascular manifestations with the picture of cor pulmonale.

The findings in the present series agree with the above conclusions. Five of the 52 cases had generalized emphysema, but these were cases who suffered from asthma or chronic bronchitis. That these latter were associated conditions, at least in the majority of cases, is shown by the fact that disappearance of the symptoms and signs took place on antibilharzial treatment in only four of 22 cases in which the effects of treatment were recorded. Any series of cases of asthma or bronchitis would probably show a similar incidence of improvement on hospitalization and symptomatic treatment.

Manifest or gross bronchiectasis was present in none of our cases. In a few, however (four of 18 cases who had bronchography), minimal dilatation was present in a subsegmental branch. As all these cases had generalized bronchitis or asthma, they are probably associated and not truly bilharzial, which fits with the pathological evidence of the non-involvement of the gross bronchi.

In the mass radiographic survey of the population of Qualioub, the results were highly significant. The incidence of pulmonary parenchymatous shadows was no higher in bilharzial individuals than in the rest of the population.

"*Bilharzial asthma.*" In the present series, 26 cases of the 57 examined had "asthma" or "asthmatic bronchitis." Of the 16 cases in which the result of treatment was recorded, improvement was noted in eight, no change in six, while in two the condition became worse. The observations were limited to the period of hospital stay. A longer follow-up would be necessary before making a final conclusion, but it is our impression that the direct role of bilharzial infection is small, as similar results are usually obtained with the usual symptomatic treatment of asthma. It is also the impression of the writers from the everyday experience of the treatment of asthma in Egyptian peasants that it is exceptional for asthma to be definitely cured after antibilharzial treatment.

It is our opinion that while bilharzial infection may be a factor in the production of asthma, its role in this respect is only of minor importance; more observation, however, with longer periods of follow-up, is necessary before a final judgment can be made.

### SUMMARY

1. The chest findings in 57 cases of bilharzia admitted to hospital have been analyzed.

2. The only finding directly attributed to the bilharzial infection was presence of ova in the sputum, which were present in three of 29 cases.

3. The following findings were possibly, but not certainly, due to bilharzia: (a) localized rales, (b) radiological shadows of a localized patchy or strandy nature, (c) asthma in which improvement occurred after antibilharzial treatment.

4. The following findings were most probably associated, and not casually related: (a) asthma or asthmatic bronchitis, in which no improvement occurred after specific treatment, (b) generalized emphysema, (c) minimal dilatation of subsegmental bronchi.

5. In a mass radiography survey of an endemic area, the incidence of parenchymatous pulmonary shadows was comparable in bilharzial and non-bilharzial subjects.

### RESUMEN

1. Se hace un estudio de los hallazgos en el tórax de 57 casos de bilarzirosis admitidos al hospital.

2. El único hallazgo directamente atribuido a la infección por bilarzia, fué la presencia de huevecillos en los esputos, que se observaron en tres de 29 casos.

3. Los hallazgos siguientes posibles, pero no ciertamente, se deben a la bilarzia: (a) estertores localizados; (b) manchas radiológicas de forma moteada o en bandas; (c) asma que mejoró después de tratamiento contra la bilarzia.

4. Los siguientes hallazgos fueron probablemente asociados y no casualmente en relación con la bilarzirosis: (a) asma o bronquitis asmática que mejoró después del tratamiento específico; (b) enfisema generalizado; (c) dilatación mínima de los bronquios subsegmentarios.

5. En una encuesta radiográfica en masas en el área endémica, la incidencia de manchas pulmonares parenquimatosas, fué comparable en los sujetos con bilarzia y sin ella.

### RESUME

1. Les auteurs ont analysé les constatations thoraciques faites dans 57 cas de bilharziose admis à l'Hôpital.

2. La seule constatation directement attribuée à la bilharziose fut la présence d'oeufs dans l'expectoration, qui ont été constatés dans trois des 29 cas.

3. Les constatations suivantes furent vraisemblablement mais pas certainement, attribuables à la bilharziose: (a) râles localisés; (b) ombres radiologiques en forme de taches ou d'infiltrats localisés; (c) asthme dont l'amélioration survint à près traitement de la bilharziose.

4. Les constatations suivantes ne furent probablement dues qu'à une association fortuite et n'ont aucun rapport causal: (a) asthme ou bronchite asthmatique dont aucune amélioration ne survint après traitement spécifique; (b) emphysème généralisé; (c) dilatation minime des bronches sous-segmentaires.

5. Dans un contrôle radiographique systématique de la zone endémique, la fréquence des ombres pulmonaires fut comparable chez les sujets atteints de bilharziose et chez les sujets indemnes.

### ZUSAMMENFASSUNG

1. Die Thoraxbefunde von 57 Fällen von zur stationären Behandlung eingewiesen Bilharzia-Erkrankungen wurden ausgewertet.

2. Der einzige direkt der Bilharzia-Infektion zugeschriebene Befund bestand in der Anwesenheit von Eiern im Sputum, die in 3 von 29 Fällen vorkamen.

3. Die folgenden Befunden waren möglicherweise, aber nicht mit Sicherheit, die Folge der Bilharzia-Infektion: (a) umschriebene Rasselgeräusch; (b) röntgenologische Verschattungen umschriebener, fleckiger oder streifiger Natur; (c) Asthma, bei dem eine Besserung nach gegen die Bilharzia gerichteter Behandlung intrat.

4. Die folgenden Befunde waren höchstwahrscheinlich verknüpft und nicht ursächlich im Zusammenhang stehend mit der Bilharzia-Erkrankung: (a) Asthma oder asthmoide Bronchitis, die sich nach spezifischer Behandlung nicht besserten; (b) ein allgemeines Emphysem; (c) minimale Dilatationen der Subsegment-Bronchien.

5. Bei Röntgenreihen-Untersuchungen war das Vorkommen parenchymatöser pulmonaler Schatten vergleichbar bei Bilharzia Kranken und bei solchen ohne diese Erkrankung.

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# SECTION ON CARDIOVASCULAR DISEASES

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## Open Heart Surgery in 1960 "A Many Splendour'd Thing"

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"The angels keep their ancient places  
Turn but a stone, and start a wing!  
'Tis ye, 'tis your estranged faces  
That miss the many-splendour'd thing."  
From "In No Strange Land"

The development of open heart procedures on a large scale during the last three years has been responsible for tremendous advances both in the techniques and in the scope of cardiac surgery. In perhaps no other field of medicine has such dramatic and fruitful progress been made. The application of a direct vision technique to congenital and acquired intracardiac defects not only has extended the technical horizons of the surgeon and the diagnostic vision of the cardiologist, but also has acted immeasurably for the benefit of the patient.

A misconception worthy of note, however, is that there is any such thing as a "standard open heart" set-up. Our "usual" technique of opening the chest via a median sternotomy incision with cannulation of the venae cavae and femoral artery is now reserved almost exclusively for the surgery of ventricular septal defects.

Direct vision surgery, for instance for aortic stenosis, or for aortic insufficiency, is accomplished necessarily by direct vision cannulation of the ostia of the coronary arteries by way of the open aorta for antegrade perfusion of the heart muscle. This was first accomplished by Dr. Charles P. Bailey<sup>\*</sup> and the author in a woman on April 18, 1958 (Fig. 1A,B). In aortic cases, drainage of the right atrium with a single large catheter (size 38 French) is preferable to individual drainage of the cavae.

For the "open" repair of mitral insufficiency at our clinic, Dr. Henry T. Nichols, the surgeon-originator, and the author employ a single catheter which is introduced into the *right ventricle* to collect the venous return from the heart, having approached it through an exclusively *left* thoracic incision. Under guidance of direct vision, sutures are placed into and through the mitral annulus fibrosus for polar cross-plication<sup>2,3</sup> (Fig. 2A,B,C). A torn leaflet may also thus be accurately repaired.

For the relief of most cases of mitral stenosis, we use this same cannulation technique and again an exclusively left thoracic incision for true "open" heart correction of the lesion. The open technique allows the

<sup>\*</sup>Thompson, Francis, "In No Strange Land," verse IV, p. 1049. *The New Oxford Book of English Verse*, Oxford Press, 1957.

addition of prosthetic material to the valve substance and permits both the removal of calcium and critically accurate division of the commissures.

At our clinic, a closed but "assisted" technique through an exclusively *right* thoracic incision is still employed for some poor risk patients with mitral stenosis. The multiperforated end of the drainage catheter used for this procedure is caused to pass through the right atrial wall, through an incision made in the interatrial septum, finally coming to lie entirely within the left atrium. The bypass is thus exclusively of the left side of the heart. This technique necessitates only a small amount of additional citrated and no heparinized blood. No oxygenator and but one pump head is needed. One femoral artery suffices for the circulatory return (Fig. 3). Should an open technique be found necessary after palpation of the valve, an oxygenator is placed in the line and the catheter is kept within the right atrium—the septum being left imperforate.

Pure pulmonary valvular stenosis in most clinics today is relieved electively by an "open" technique using hypothermia (85°F). Such a method allows full and accurate opening of the congenitally fused commissures with far less chance of producing valvular insufficiency than the closed "Sellors-Brock" type of operation. The fall in the gradient post-operatively when this technique is employed is much greater than with the closed technique (Fig. 4). This is one lesion of the heart that uniformly can be alleviated within the permissible hypothermic time limit.

Similarly, congenital aortic stenosis often can be operated satisfactorily with inflow occlusion and hypothermia alone in children; in young adults extracorporeal circulation is easier and safer.

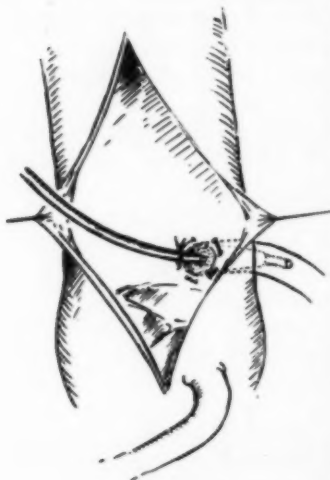


FIGURE 1A

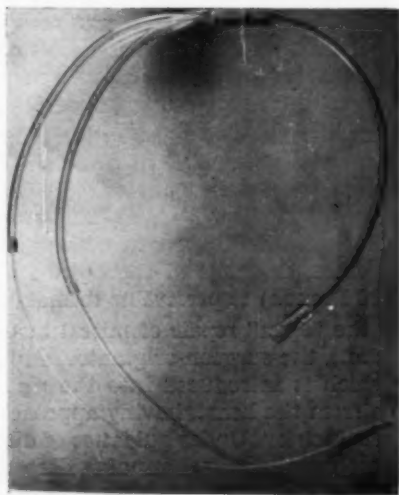


FIGURE 1B

FIGURE 1A: Small plastic catheters are inserted into the coronary ostia through the open aorta. They are secured in place by a tiny pursestring which also prevents leakage around the foam rubber cuff.

FIGURE 1B: Photograph of the actual catheters as designed by Nunez and obtainable from the Abbott Laboratories, North Chicago, Illinois. These catheters are in routine use on all acquired cases of aortic stenosis and insufficiency.

For the repair of complicated congenital defects such as the tetralogy of Fallot, the author and his associates have been employing a pump-oxygenator and direct cooling of the extracorporeal blood with a heat-exchanger to effectuate extreme hypothermia (to 10-15°C). This developmental combination of bypass and hypothermia with intermittent circulatory interruption allows a prolonged operative period with a "dry" field. The patient may be temporarily exsanguinated into the extracorporeal circulation to facilitate an even more rapid development of a dry field. This method has been simpler in our hands than the use of deep hypothermia with extracorporeal cooling but *without* an oxygenator as described by Drew.<sup>4</sup>

The technic of repair of ventricular defect, aortic stenosis, and mitral stenosis are described in detail to illuminate the differences in the "open" approach to these three lesions.

### *The Open Repair of Ventricular Defect*

#### *Indications:*

If a patient over one year of age has an uncomplicated ventricular septal defect with (a) net left-to-right shunt of over one liter, (b) no peripheral cyanosis at rest, and (c) a right ventricular pressure appreciably less than his systemic arterial blood pressure, he is felt to be a candidate for the "standard operation." If he has almost equal pressures on the two sides of the heart with a marked net left-to-right shunt, a pulmonary constriction operation, or closure of the defect with a pros-

TABLE 1

The choice of incisions, techniques, cannulations, and types of bypass for various open, and assisted heart surgical procedures.

<i>Lesions</i>	<i>Approach</i>	<i>Surgical Technique</i>	<i>Type of Bypass</i>	<i>Cannulation</i>
Mitral stenosis	Left lateral thoracic 5th interspace incision	A. Neostrophingic <sup>6,7,8</sup> mobilization of the valve—both digital and instrumental technique at valvular and subvalvular levels <i>under direct vision</i> . Removal of clots and loose calcium.	Total bypass with pump-oxygenator. Usually no hypothermia.	Right ventricle (pulmonary artery obstructed) to femoral artery or descending thoracic aorta.
				Right atrium to femoral arteries.
Mitral insufficiency	Left posterolateral 6th interspace incision.	B. Annulus plication, <sup>3</sup> or method below depending on pathology.  C. Repair of torn or cut leaflet by suture. <sup>9</sup>  D. Ivalon buttress support of mitral leaflet if there is an actual dearth of valvular substance, <sup>9</sup> or dacron cloth sleeve repair of ruptured chordae etc.	Open with pump-oxygenator.  Open with pump oxygenator.  Open with pump oxygenator.	Right ventricle to femoral artery.  Right ventricle to femoral artery.  Right ventricle to femoral artery.

thetic (one-way) valve is performed. If there is an equalized *shunt*, lung biopsy is obtained preoperatively. Depending upon the pathologic appearance of the pulmonary vessels as interpreted by a pathologist with considerable experience in this field, a decision is reached as to whether the vessel damage is significantly reversible, or whether there is no pulmonary vascular reserve.

In patients under one year of age operation often may be deferred unless impending heart failure as evidenced by pulmonary congestion and repeated pneumonia is present, in which case pulmonary constriction<sup>11</sup> is carried out.

### Operation—The Thoracic Incision

The operation for closure of uncomplicated ventricular defects is performed as follows: An absolutely straight and midline incision is made over the sternum. This is important not only for cosmetic reasons but also to insure that the mediastinum below the sternum will be entered without opening either pleural space. The incision is extended only for a limited distance above the sternum as bleeding from the vascular tissue of the neck can be a problem after open heart surgery. The lower extremity is extended well beyond the xiphoid cartilage. The latter is removed, to facilitate postoperative drainage of the pericardium, if neither pleural cavity is drained.

The sternum is cut through vertically in the midline with an electric (non-rotating) Stryker saw. The Lebsche knife and Shumacher shears are avoided in order to obtain an even and straight incision without fracture of the tables, excessive hemorrhage, or perforation of the pleura.

The anterior mediastinal tissues are gently separated by wiping with gauze until the anterior aspect of the pericardium is exposed. The pericardium is opened longitudinally.

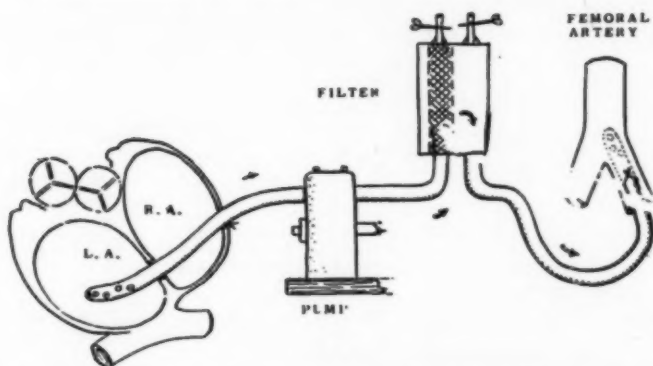


FIGURE 3: This simple yet effective bypass of the left ventricle and mitral valve consists only of a pump and a filter. A small amount of ordinary citrated blood is sufficient to prime the system which is quickly assembled. This bypass allows careful, unhurried instrumentation of the valve under continuous digital guidance. Hypotension during surgery is reduced and deaths from this cause may be eliminated. This technique for supporting the circulation during surgery for mitral stenosis is now only used when sufficient blood of a rare type is not available or when the patient is too weak (rare) to withstand full "open" surgery.

### Monitoring

A small plastic catheter is inserted in the aorta and is connected to a manometer to record the arterial pressure before, during, and after bypass. A polyethylene catheter for measurement of the venous pressure is inserted into the inferior (or both) vena cava. Over-replacement of blood is thus avoided.

TABLE 2

Lesion	Approach	Surgical Technique	Type of Bypass	Cannulation
Aortic stenosis.	Midline vertical sternotomy.	"Sculpturing" of the valve. Subtotal debridement and excision of calcium; commissurotomy. <sup>10</sup>	Open, antegrade coronary cannulation, perfusion by a coronary perfusion pump, and conventional pump oxygenator, with mild hypothermia.	Rt. atrium single catheter drainage to femoral arteries. Separate catheters into each coronary ostium. Two <sup>11</sup> low-pressure venous suction tips for cor. sinus return—one placed thru apex of left ventricle to obviate air embolism.
Aortic insufficiency.	Midline vertical sternotomy.	(Depending upon pathology) 1. Bicuspidization by excision of a coronary cusp and aortic wall plication. <sup>8</sup> 2. Bicuspidization by suture of two adjacent leaflets together. <sup>8,11</sup> 3. Leaflet lengthening by calcium excision and "unrolling" of edge. <sup>12</sup> 4. Leaflet lengthening by use of prosthetic "patch." 5. "Patching" of fenestrations of cusps.	Open with antegrade coronary perfusion, and pump oxygenator as above.	Rt. atrium single catheter drainage to femoral arteries. Separate catheters into each coronary ostium.

### *Cannulation*

The superior end of the pericardial incision is extended to the utmost limit of the attachment of the pericardium about the aortic arch. Umbilical tapes are placed around the venae cavae using a Rumel "D" clamp.

Heparin is administered intravenously in dosages of 1.5 mgm. per kilogram of bodily weight. If femoral catheterization is chosen, 10 minutes are permitted to elapse, then the common femoral arteries (thru a transverse incision in skin and artery) and the two venae caval catheters are cannulated separately by plastic catheters stiffened by blunt malleable obturators (Fig. 5AB). Our favored technique for fixation is to place a separate purse-string suture (atraumatic 3-0 Nylon) on each surface (or "edge") of the right atrial appendage. The catheters are introduced individually through separate incisions made within the confines of the pursestrings. The pursestring sutures are tied and then each is wound twice around the appropriate catheter and retied. The catheters are connected with the appropriate lines from the bypass equipment.

Finally, the clamps are removed from the femoral catheters and then first from one vena caval catheter and then from the other (to avoid the possibility of any entrapped air entering the right atrium), and the pumps are started gradually. When a flow greater than 50 cc/kg. has been attained, and a satisfactory balance and blood pressure have been established, the umbilical tapes are tightened around the vena caval catheters. An additional pump is now turned on which is connected to a Russell suction tip for "low-pressure" suction.

In an increasing number of patients but especially in very small infants with hypoplastic femoral arteries, arterial cannulation may be quickly and easily carried out by making an incision into the ascending aorta near the arch.

### *The Cardiac Incision*

A longitudinal incision is made between stay sutures of medium cotton from the region of the pulmonary valve to the ventricular apex staying within the "avascular" region and taking care not to interrupt any large coronary arterial branches.

The defect is exposed by emptying the ventricle with the "low-pressure" suction tip and without cardiac arrest. If a small degree of aortic insufficiency is present, 3 to 4 minute periods of aortic occlusion by a non-crushing clamp (Fig. 6) is employed to obtain a clear view. Direct suture is used if the defect is less than one cm. in maximum diameter. If larger than this, an Ivalon patch reinforced with incorporated Teflon mesh is sutured in place. If it is felt desirable or necessary to maintain the possibility of a temporary right-to-left shunt postoperatively, an "artificial foraminal valve" is used to patch the defect. Care is taken to avoid the common conduction bundle which usually lies between 9 and 6 o'clock on the dorso-caudal portion of the margin of the defect. In this area sutures should be passed parallel to the margin of the defect and not perpendicular to it in order to obviate any possibility of enclosing even a part of the bundle. The ventricular incision is then closed with a running over-and-over suture of non-absorbable medium suture reinforced by a continuous everting suture of the same material. If there is still leakage at any point, additional interrupted mattress sutures should be placed.

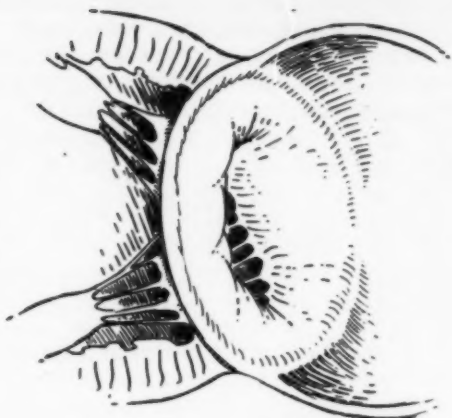


FIGURE 2A

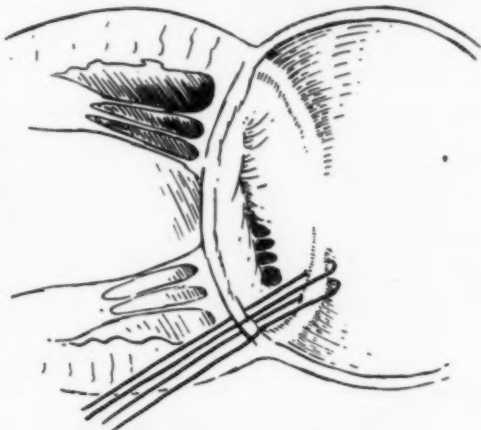


FIGURE 2B

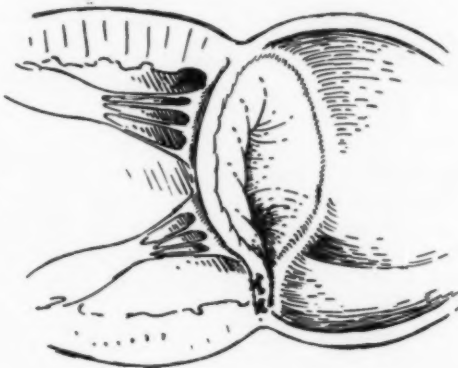


FIGURE 2C

FIGURE 3: A. Mitral insufficiency is usually manifest as a failure of the leaflets to coapt at the postero-medial or "right" commissure. B. Under direct vision sutures are placed in such a manner as to encircle part of the annulus fibrosus on each side of the postero-medial pole of the valve. C. When these sutures are tied the annulus assumes a snow-shoe shape and the valve leaflets are made to coapt by the approximation of their bases. Although the illustration shows the sutures tied outside the heart, with the present technique, the knots (of No. 2 Nylon) are now inside the atrium. (Nichols, H. T., and Bailey, C. P.: "Ch. on Mitral Regurgitation," *Encyclopedia of Thoracic Surgery* [Derra] Springer-Verlag, Berlin, 1956.)

The coronary return is allowed to flood the field as the heart wall is closed. This safeguards against air embolism. Carbon dioxide gas<sup>19</sup> may be led into the chest during the closure to displace any air which might be entrapped within the irregular recesses of the heart.

When the heart has resumed beating with full vigor, the rate of bypass is reduced. The heart is allowed to gradually take over the entire circulation again. Calcium salts, digitalis, or adrenalin may be given at this time if necessary to increase the tone and vigor of myocardial contraction.

### *Decannulation*

When an adequately stabilized circulation has been obtained, the caval and femoral catheters are withdrawn and polybrene<sup>20</sup> is given to restore coagulability to the blood. It is given in dosage of two-to-one to neutralize the heparin. Hemostasis of the soft tissues and the sternal periosteum is painstakingly obtained and the upper three-fourths only, of the pericardial incision is closed with interrupted sutures.

A multi-perforated twin (Lloyd) catheter (Fig. 8) is placed between the diaphragm and the heart. A warm sterile saline flush is carried out intermittently for 48 hours through one limb (in one, out the other) of this catheter to minimize the likelihood of intra-pericardial clotting and to prevent tamponade. The other limb is connected to a water seal bottle with gentle controlled (10-20 cm H<sub>2</sub>O) suction. The cut sternal margins are approximated after 4 sutures of braided wire have been placed using a strong curved needle to perforate the sternum itself.

TABLE 3

<i>Lesion</i>	<i>Approach</i>	<i>Surgical Technique</i>	<i>Type of Bypass</i>	<i>Cannulation</i>
Aortic and mitral stenosis combined.	Midline vertical sternotomy.	Open operation for both valves. Mitral approached thru septum.	Conventional bypass. Antegrade cannulation and perfusion of coronary arteries.	Vena cava to femoral artery or aorta.
Aortic stenosis and aortic insufficiency.	Midline vertical sternotomy.	Technique as above.	Conventional bypass and antegrade coronary perfusion.	As in isolated aortic stenosis.
Mitral stenosis with significant mitral insufficiency.	Left posterolateral incision.	As for each separate lesion.	As described for mitral insufficiency.	As described for mitral insufficiency.

The subcutaneous tissues are closed with interrupted very fine wire. The skin incision is repaired with a continuous subcuticular pull-out wire. A heavy mattress suture is placed (but not tied) for subsequent closure of the opening which will be left when the Lloyd catheter is removed. If either pleural space has been violated, the usual type of intercostal drainage with a water-seal system is instituted.

### *The Relief of Aortic Stenosis by Open Heart Surgery*

Patients with acquired aortic stenosis with a significant gradient (over 50 mm. Hg.) across the aortic valve are considered surgical up to the age of 70 unless there is associated advanced coronary artery

disease. In suspicious cases in the older age group, catheter aortography (Fig. 8A) is employed to visualize the coronary arteries definitely. The presence of concomitant aortic insufficiency can be quantitated simultaneously by this study (Fig. 8A+B) and is not necessarily a contraindication to surgery. Such insufficiency responds surprisingly well to cusp lengthening and unrolling following removal of calcium.

An effective operation for the proper relief of calcific aortic stenosis is not easy and requires a meticulous "cuspi-cure" of the valve. A vertical midline incision is made as described above. After heparinization the tip of a single large (#40 French) multiperforated catheter is placed within the right atrium and femoral (or direct aortic) cannulation is carried out.

It should be noted that the blood is collected from the atrium in this system by siphon gravity drainage. The pulmonary artery is encircled by an umbilical tape which is tightened when complete bypass flow rates are attained.

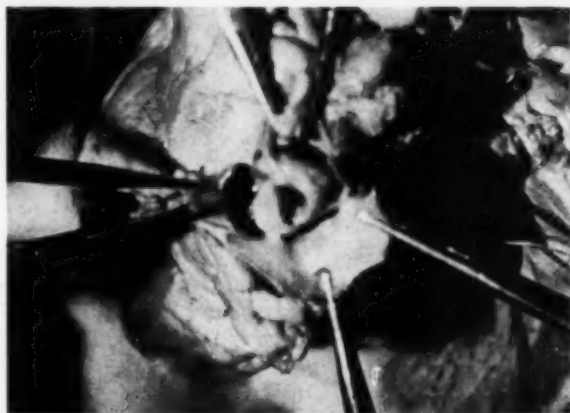


Figure 4A

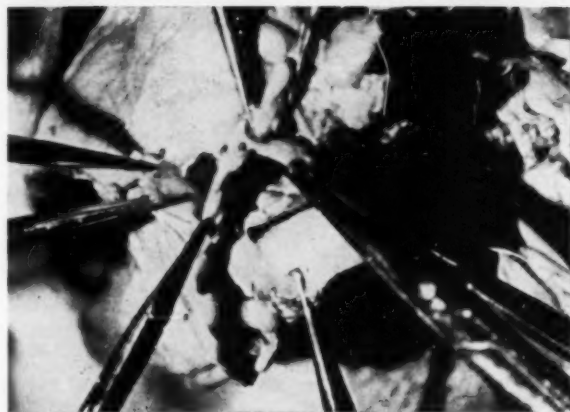


Figure 4B

**FIGURE 4A,B:** Photographs of a valve with congenital pulmonary stenosis before and after commissurotomy. Note the easily recognized lines of the fused commissures. (Bailey, C. F., Musser, B. G., and Morse, D. P.: *Am. J. Cardiol.*, 4:147, 1959.)

TABLE 4

Lesion	Approach	Surgical Technique	Type of Bypass	Cannulation
Atrial septal defect 1) secundum type	Vertical midline incision	Open repair by suture or patch.	Conventional pump oxygenator.	Conventional venal caval and femoral arterial or aortic cannulation
2) primum type	Same	Open-insertion of prosthesis; repair of associated mitral or tricuspid insufficiency by leaflet suture.	Pump oxygenator and hypothermia severe (to 12°C) with circulatory arrests up to 20 minutes	Separate vena cava catheters. Effluent limbs into femoral arteries. Left persistent superior vena cava may require obstruction or cannulation.
3) with anomalous pulmonary vein drainage	Same	1) either by "double barreling" <sup>11a</sup> of superior venae cava, or vein transfer to defect.	As above	2) As above
4) with nearly balanced shunts	Same	Insertion perforated prosthesis or artificial foraminal valve of Ivalon which will permit a temporary right to left shunt only. <sup>13</sup>	2) As above but normothermic usually.	2) As above

The aorta is then cross-clamped with a fenestrated non-crushing clamp (see Fig. 6) and a proximal longitudinal aortotomy is made between stay sutures of 3-0 cotton. The low pressure suction tip is passed through the diminutive valve opening to remove the bronchial venous and left ventricular coronary blood return.

The left coronary ostium is pursestringed from the inside with a 3-0 nylon suture swedged on a special curved needle,\* and a special small plastic (Nunez-Tello\*\*) catheter with a rubber foam cuff (see Fig. 1B) is inserted and fastened in place. The right coronary catheter is guided

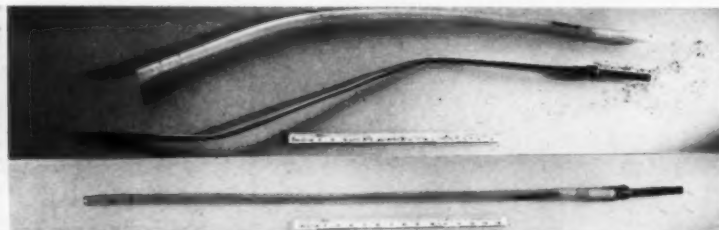


FIGURE 5A (upper): A blunt malleable obturator for a venous catheter. This device, developed by Dr. Edward Fitch at our clinic, facilitates bloodless introduction of soft plastic catheters thru the atrial appendage into the mouth of the venae cavae. FIGURE 5B (lower): Obturator within catheter. (Fitch, E. A., and Bailey, C. P.: *J. Thor. Surg.*, 37:660, 1959.)

\*Obtainable from Ethicon Suture Division, Johnson and Johnson, New Brunswick, New Jersey.

\*\*Obtainable from Abbott Laboratories, North Chicago, Illinois.

TABLE 5

Lesion	Approach	Surgical Technique	Type of Bypass	Cannulation
Ventricular septal defects	Vertical midline incision	Open technique. Primary closure if 1 cm. or less in size.	Pump oxygenator (intermittent aortic occlusion if aortic insufficiency is present) no arrest.	Individual vena caval catheters through wall of right atrium; femoral artery or direct aortic cannulation.
1) uncomplicated				
2) less than 12 months old with severe pulmonary hypertension	Left anterior 3rd intercostal space incision.	Closed technique. Constriction of pulmonary artery to produce relative pulmonary stenosis using continuous pressure recording control. <sup>13</sup>	None	None
3) severe pulmonary hypertension	Vertical midline incision.	Open technique. Closure of defect with prosthetic foraminal valve.	Pump oxygenator as above.	As in (1)
4) with minimal pulmonary stenosis—"acyanotic tetralogy"	Vertical midline incision.	Open technique. Complete correction see ventricular septal defect and pulmonary stenosis.	Pump oxygenator as above.	As in (1)
Cyanotic tetralogy of Fallot:	(Two stage operation)	Limited pulmonary or partial infundibular resection	None	None
1) with very small pulmonary and/or outflow tract	Stage I Left anterior 4th intercostal space incision.	(Brock) or Blalock type operation.		
	Stage II As in acyanotic tetralogy above.	As in acyanotic tetralogy.	Pump oxygenator and hypothermia.	As in acyanotic tetralogy.
2) cyanotic tetralogy of Fallot with fair to normal sized pulmonary artery and/or outflow tract	Vertical midline incision.	Open technique. Infundibular ridge may be used as a flap to close defect.	As above	As above

into the appropriate ostium, and is fixed in place by a suture which is passed around the coronary artery and is tied over a tiny sponge to facilitate later release.

#### *"Cuspi-cure" or Sculpturing of the Aortic Cusps*

After the coronary perfusion has been started, the heart soon regains its forceful beat which has been lost due to anoxia. Ventricular fibrillation may have developed or may be induced electively at this time. The aortic valve is slowly trimmed or "sculptured" using scissors or special rongeurs. The ventricular cavity may be packed with a long piece of half-inch gauze to prevent loss of any small calcific fragments into this chamber. The commissures are carefully opened with knife blade, guillotine knife, or scissors. Sometimes only one or two commissures, usually the anterior and the right postero-lateral, may be opened fully. Opening the commissure between two cusps which have lost their deep cup-like shape may allow one of these (if shelf-like) to invert. Nearly always,

by careful trimming (cuspi-cure), at least one cusp can be fully mobilized and thinned sufficiently to become fully pliable (Fig. 9). It is important not to work on both the inside of a cusp and on the "channel" side in the same area as fenestration might then occur." Any mass of subvalvular calcification especially if beneath the commissures, should be removed.

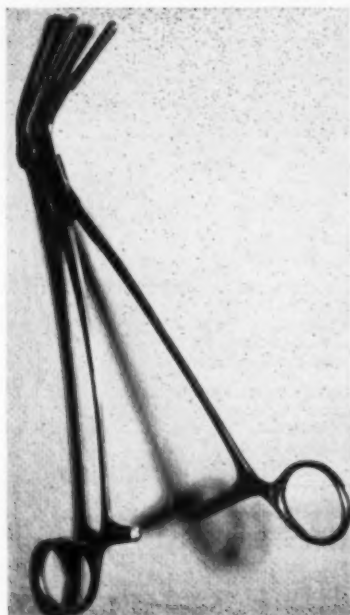


FIGURE 6



FIGURE 7

FIGURE 6: Photograph of the fenestrated aortic cross-clamp (the "Duckbill") which is designed for multiple applications without injury to the aortic intima or surrounding structures. This and all other instrumentals described are obtainable from Pilling Co., Philadelphia.

FIGURE 7: Photograph of Lloyd double catheter which is inserted within the pericardium and beneath the heart for the first 48 hours postoperatively. Irrigation drainage of this type allows preservation of the integrity of both pleural cavities thus lessening the incidence of postoperative pulmonary complications.

TABLE 6

Lesion	Approach	Surgical Technique	Type of Bypass	Cannulation
Pure pulmonary stenosis (valvular)	Vertical midline incision.	(Open) commissurotomy	Inflow occlusion hypothermia 85°F.	None
Same (infundibular)		(Open) resection	Conventional bypass. Normo-thermic.	Vena caval and femoral artery.
Congenital aortic stenosis.	Vertical midline incision.	(Open) commissurotomy or relief of subvalvular stenosis	Inflow occlusion hypothermia 85°F. or Conventional bypass.	None or Vena caval and femoral artery.

### *Closure of the Aortic Incision*

The aorta is subtotally closed with 4-0 atraumatic Deknatel sutures using a continuous everting mattress sutured reinforced by an over-and-over technique, and commencing at the cardiac end of the incision.

When the aortic wound has been closed to such an extent that the remainder can be clamped, the left coronary catheter is cut loose within the aorta and is removed. The sutures about the right catheter are released and it is removed. The heart and the aorta are allowed to fill with blood by releasing the tape around the pulmonary artery and partially blocking the drainage from the left atrial catheter. After all air has been forcefully ejected by manual compression of the ventricles the remaining open portion of the incision is closed with a Beck aortic or a Satinsky clamp with non-slip jaws and the distal aortic cross-clamp is released.

To avoid air embolism from any small bubbles trapped in the trabeculae of the left ventricle the author now places a small (No. 10 French) catheter in the apex of the left ventricle through the wall. This is left in place while the aortic incision is closed allowing continuous decompression of the left ventricle. Finally, when the heart is beating well, the apex is lifted and the catheter slowly withdrawn.

After cessation of bypass the various catheters are removed and the vessels are repaired. Then, after heparin neutralization, the chest is closed as described in respect to ventricular septal defect repair.

TABLE 7

<i>Lesion</i>	<i>Approach</i>	<i>Surgical Technique</i>	<i>Type of Bypass</i>	<i>Cannulation</i>
Coronary artery disease	Vertical midline or left lateral incision (depending on location of lesion).	Endarterectomy <sup>17</sup> or thrombo-endarterectomy.	Bypass and extreme hypothermia (10-15°C) induced by heat exchanger.	Vena cavae to femoral artery.
a) localized block (by coronary visualization) <sup>18</sup>				
b) generalized disease	Left lateral incision 6th interspace.  or Supine through bed of 4th costal cartilage.	Internal mammary implantation (Vineburg)	None	None
		Poudrage.	None	None

### *The "Open" Relief of Mitral Stenosis*

The closed classical operations for mitral stenosis are satisfactory in only 70 per cent of the cases.<sup>21</sup> The frequent co-existent mitral insufficiency, atrial thrombosis, and extreme fibro-calcific transformation of the valve have led to this conclusion by many.

The operation can be better performed under direct vision as described below when any of the above conditions are suspected. The majority of the author's patients are now so operated in Philadelphia.

With the patient supine the femoral arteries are isolated—they are difficult to dissect in the lateral position—and the patient is then turned

with the left side up. Through a wide 5th interspace incision the pericardium is opened and heparin given.

The venous drainage catheter is placed in the right ventricular outflow tract through a pursestring of No. 2 Nylon. The arterial limb of the pump is connected either to the femoral artery, or directly into the descending thoracic aorta. After the bypass is established, the pulmonary artery is clamped (See Fig. 6), and the atrium opened along the atrio-ventricular ring.

As the valve is opened under direct vision at each commissure with special scissors, it is held open by a ventricular suction tip to prevent air embolism.

The valve incision is extended in a semicircle to insure mobility of the septal (aortic) leaflet. Any regurgitant element is repaired. Any sub-valvular cross-fusion is separated.

As the atrium is closed a suction vent is left in the left ventricle to avoid air embolism and to ensure continuous decompression as the heart regains its vigor. After these catheters are withdrawn from the heart and arteries, polybrene is administered (*vide supra*) and the chest closed in the usual manner.

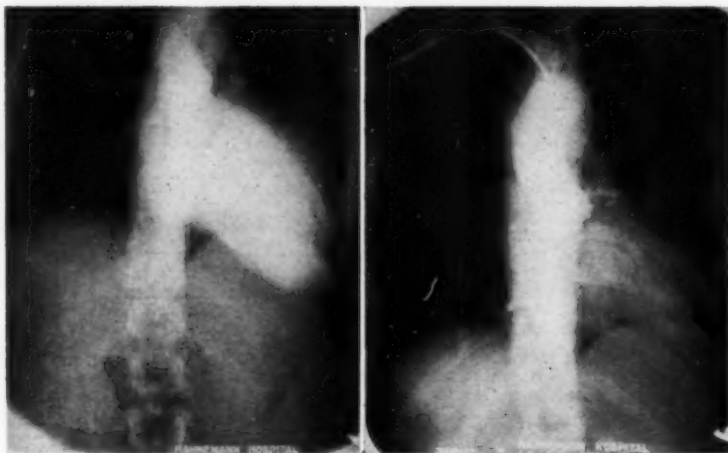


FIGURE 8A

FIGURE 8B

FIGURE 8: These are two comparable aortograms on the same patient before and after surgery for aortic valvular insufficiency. Fig. 8A (6-3-58) represents the sixth film in a series taken at half-second intervals following the injection of a radio-opaque dye (90% Hyopaque) into the ascending aorta by a long needle introduced through the suprasternal notch in the neck. The film shows massive regurgitation of the dye into the dilated left ventricle. Fig. 8B (7-16-58) was taken postoperatively and demonstrates the absence of any significant regurgitation. The left coronary artery is well visualized. Bailey, C. P., Musser, B. G., and Morse, D. P.: *Am. J. Cardiol.*, 4:147, 1959.

#### SUMMARY

Open heart surgery, therefore, is not simple. Many lesions of the heart require special techniques and individual bypass arrangements. The challenge of the future lies in the development and perfection of the safest methods of utilizing this "magnificent thing." Our present techniques which are subject to slow but continuous change are shown in the accompanying table.

The basic techniques now employed in three such common and different operations as ventricular septal defect, acquired (calcific) aortic stenosis, and major mitral stenosis, are appended in outline form. The new principle of correction of aortic insuf-

Figure 9A

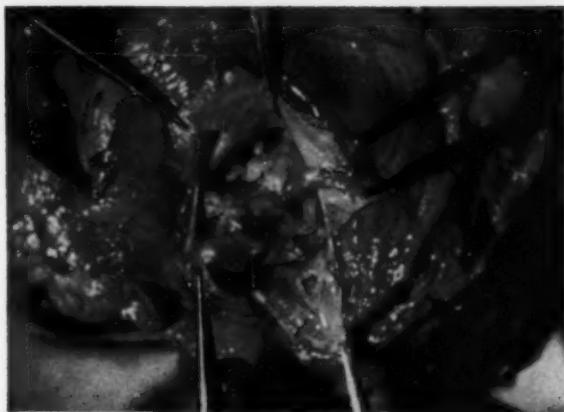


Figure 9B



Figure 9C



FIGURE 9A: Photograph of an extremely calcified and immobile aortic valve.

FIGURE 9B: Photograph of same valve after "cuspi-cure."

FIGURE 9C: Photograph of same valve illustrating restored mobility. (Bailey, C. P., Musser, B. G., and Morse, D. P.: *Am. J. Cardiol.*, 4:147, 1959.)

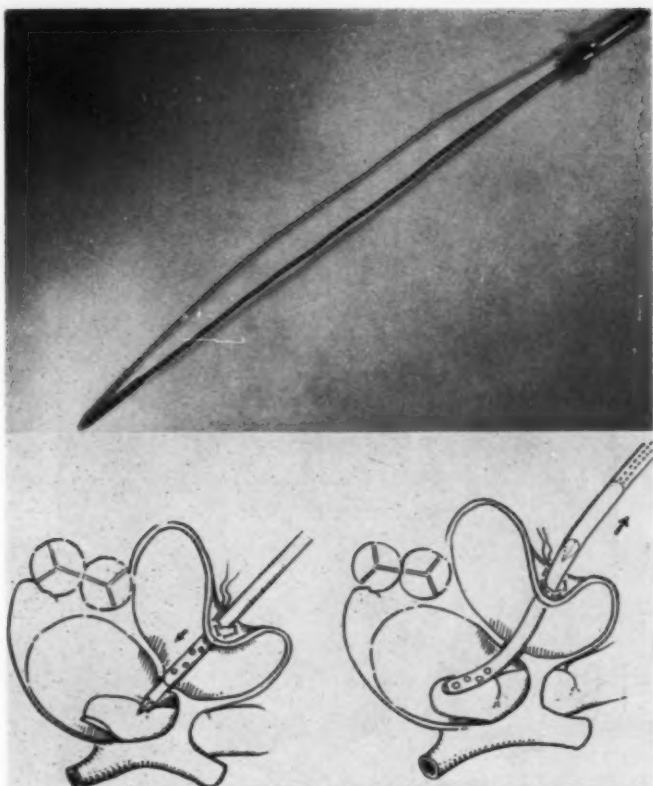


FIGURE 11A: Photograph of the malleable sharp, trocar-pointed obturator employed to introduce a venous catheter without preliminary incision not only through the right atrial wall, but also through the interatrial septum. The edge of the catheter tip must be beveled to allow easy introduction. This sharp pointed obturator is also useful in cannulating the pulmonary artery through the right ventricular outflow tract when using the "autogenous lung" system of circulatory bypass.

FIGURE 11B: Diagrams of the insertion of the left atrial catheter under digital guidance for closed, "assisted" mitral bypass.

ficiency by removal of the non-coronary cusp or by leaflet suture<sup>5</sup> is accomplished with a bypass technique similar to that used for aortic stenosis.

#### RESUMEN

La cirugía a corazón abierto no es sencilla. Muchas lesiones del corazón requieren técnicas especiales y arreglos individuales de las desviaciones. El problema para el futuro radica en el desarrollo y perfeccionamiento de los métodos más seguros para utilizar, es "cosa de mucho esplendor." Nuestras técnicas actuales que se sujetan a cambio lento pero continuo se muestran en el cuadro que se acompaña.

Las técnicas básicas ahora empleadas en tres de las operaciones comunes y diferentes, tales como defecto de tabique ventricular, estenosis aórtica calcificada adquirida y estenosis mitral mayor, se agregan en forma de esquema.

El principio nuevo de la corrección de la insuficiencia aórtica por la resección de la cúspide no coronaria o por sutura de la hojilla, se lleva a cabo por una técnica de desviación similar a la usada en la estenosis aórtica.

#### RESUMÉ

La chirurgie à coeur ouvert n'est pas simple. Beaucoup de lésions du coeur nécessitent des procédés spéciaux et des adaptations individuelles de la circulation extracorporelle. La lutte de l'avenir réside dans le développement et le perfectionnement des méthodes les plus sûres d'utilisation de cette "chose pleine de promesses." Nos

techniques actuelles qui sont l'objet de modifications lentes mais continues sont montrées dans le tableau joint.

Les techniques de base maintenant utilisées dans trois opérations tellement banales et différentes telles que la perforation de la paroi ventriculaire, la sténose aortique acquise (calcifications) et la grande sténose mitrale, sont annexées sous forme d'esquisse. Le nouveau principe de la correction d'une insuffisance aortique par l'exérèse de la pointe dépourvue de coronaire ou par suture de la valve est pratiqué grâce à une technique de cœur artificiel semblable à celle utilisée pour la sténose aortique.

#### ZUSAMMENFASSUNG

Die Chirurgie des offenen Herzen ist nicht einfach. Viele Herzbefunde verlangen eine besondere Technik und individuelle Anordnung für den Kurzschluß. Die Forderung der Zukunft liegt in der Entwicklung und Vervollkommenung der sichersten Methode der Nutzbarmachung dieser Gabe. Unsere augenblickliche Technik wird in der beigefügten Tabelle gezeigt und ist einer allmählichen, aber ständigen Umwandlung unterworfen. Das Grundlegende operative Vorgehen, wie es jetzt angewandt wird bei eisenso häufigen und verschiedenen Eingriffen wie Kammercheidewanddefekt, erworbene (verkalkte) Aortenstenose und größere Mitralstenose ist im Überblick beigefügt.

Das neue Prinzip der Korrektur der Aorteninsuffizienz durch Entfernung einer bestimmten Klappe wurde durch Segelnaht mit der Kurzschlußtechnik ausgeführt, ähnlich der der für die Aortenstenose gebräuchlichen.

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# Supraventricular Tachycardia in Acute Myocardial Infarction\*

## Review of the Literature and Report of Five Cases

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Supraventricular tachycardia as an arrhythmia occurring in acute myocardial infarction is uncommon, and is rare when digitalis overdosage is excluded as a precipitating factor. Freiermuth and Jick<sup>1</sup> reported eight cases of paroxysmal atrial tachycardia with 2:1 block associated with myocardial infarction in 16,481 electrocardiograms on 8,147 patients reviewed particularly for the study of paroxysmal atrial tachycardia with A-V block. Of these, three were related to digitalis intoxication, three occurred without digitalis overdosage, and no mention was made of digitalis in the other two cases in their study. Askey<sup>2</sup> reviewed 1,247 cases of myocardial infarction in Los Angeles County Hospital specifically for atrial tachycardia in myocardial infarction and found only five with supraventricular tachycardia as against 84 with atrial fibrillation, 20 with atrial flutter, and 14 with paroxysmal ventricular tachycardia. Three of the supraventricular tachycardias (cases 3, 4, and 5) were definitely related to large doses of digitalis, consisting of 2.2 gm. in 31 hours, 1.6 gm. in 24 hours, and 1.9 gm. in 24 hours respectively, given immediately before the onset of the tachycardia.

The five cases of Askey and nine other cases<sup>1,3,4,5</sup> reported under various subjects, together with our five cases, were tabulated for comparison (Table 1). Twelve other cases of supraventricular tachycardia,<sup>7,9,14</sup> including the two of Freiermuth and Jick, were omitted from the table because of insufficient data. The 19 tabulated cases and the 12 untabulated cases thus constitute a total of 31 instances of supraventricular tachycardia found in a heterogeneous group of at least 2,500 cases of myocardial infarction.

We found only five patients with supraventricular tachycardia in a review of the last 360 consecutive cases of acute myocardial infarction admitted to White Cross Hospital. Only one (case 3) was related to digitalis intoxication. Cases 1 and 2 were reverted to sinus rhythm with Cedilanid-D® (deslanoside), while case 5 developed ventricular fibrillation 10 to 15 minutes after 1.2 mg. of Cedilanid was given intravenously. Three had tachycardia with their first attack of myocardial infarction and two developed tachycardia only with their second attack. Case 5 is unusual in that both attacks of coronary occlusion were accompanied by supraventricular tachycardias of 280 and 300 per minute, respectively; while an attack of coronary insufficiency in the interim between the two acute coronary occlusions did not precipitate any tachycardia.

### Case Histories

1. C.C., a 71 year-old white woman, hypertensive for 10 years and with angina pectoris for five to seven years, was admitted in acute pulmonary edema on October

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Figure 1A

Figure 1B

Figure 1C

Figure 1D

Figure 1E

Figure 1F

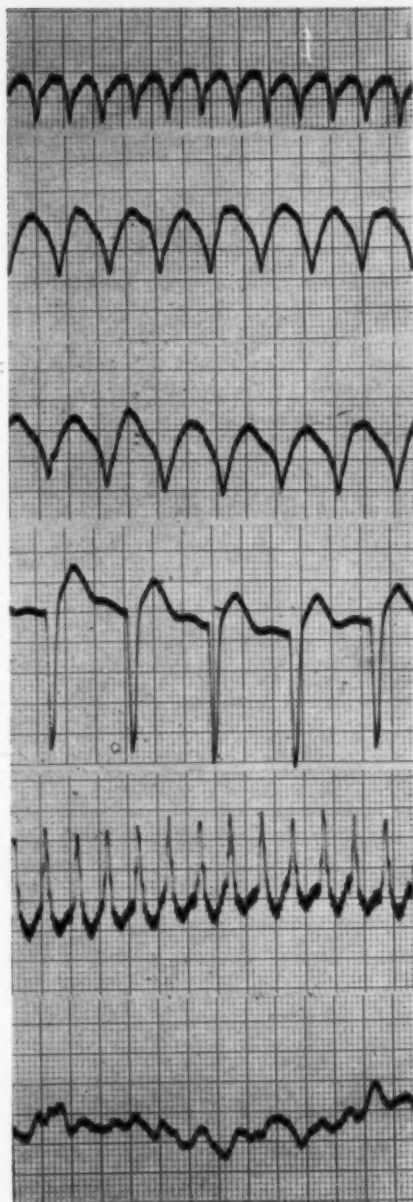


FIGURE 1. Case 5: Tracings from A to D are samples of a continuous tracing of Lead V. A: Supraventricular tachycardia with a ventricular rate of 273 per minute before beginning treatment with procaine amide intravenously. B and C: Note progressive widening of the QRS during intravenous injection of procaine amide. D: Reversion to sinus rhythm with a rate of 107 per minute after 2800 mg. of procaine amide intravenously.

Tracings E and F are continuous tracings of Lead II. E: Supraventricular tachycardia with a ventricular rate of 300 per minute taken before Cedilanid was given intravenously. F: Ventricular fibrillation occurring after a total dose of 1.2 mg. of Cedilanid intravenously. The transition to ventricular fibrillation was sudden with no noticeable prefibrillatory phase in the continuous electrocardiogram.

27, 1958 at 2 a.m. Her blood pressure was 118/70, respiration 36, and pulse 120. She was given aminophylline 0.5 gm., Mercuhydrin® 2 cc., and Cedilanid 0.8 mg. intravenously, in the emergency room. At 4 a.m. and at 9 a.m. of the same day, she received 0.4 mg. of Cedilanid. In the next 48 hours, she received 1.25 mg. of digoxin with clearing of her pulmonary edema. At 8 a.m. on October 29, 1958 she developed supraventricular tachycardia with a ventricular rate of 158 per minute. She was then given Cedilanid 0.4 mg. intravenously with reversion to sinus rhythm in about 10 minutes. In the afternoon of the same day, supraventricular tachycardia recurred with spontaneous reversion to sinus rhythm while the electrocardiogram was being taken. On October 30, 1958 she received digoxin 0.25 mg. at 9 a.m., and Cedilanid 0.4 mg. at 3 p.m. and at 12 midnight, when she developed a grossly irregular rhythm diagnosed by auscultation as atrial fibrillation. From October 31, 1958 she was maintained on digoxin 0.25 mg. daily with no recurrence of atrial or nodal tachycardia. Serial electrocardiograms showed a septal infarction with involvement of the anterior and posterior surfaces. She remained compensated until discharged.

*Comment:* As far as could be ascertained this was the initial attack of myocardial infarction of this woman. The fact that the tachycardia reverted to sinus rhythm after additional doses of Cedilanid would seem to indicate that this tachycardia was not caused by the original digitalization.

2. L.A., a 58 year-old white man with a history of posterior myocardial infarction in 1947 was admitted with sudden onset of tachycardia and precordial pain on October 29, 1958. The electrocardiogram showed a supraventricular tachycardia with a rate of 188 per minute. Carotid massage and eyeball pressure failed to terminate the tachycardia; therefore, Cedilanid 1.2 mg. was given intravenously and in 10 minutes the rhythm reverted to normal sinus. Serial electrocardiograms showed T wave changes in II, III, and AVF, compatible with a new posterior infarction. Serum transaminase determinations were 55, 50, 46 units (normal less than 40 units) done at 12 hour intervals from the time of admission. The rest of the hospital course was benign and he was discharged improved.

*Comment:* The rapid termination of the tachycardia possibly prevented more severe damage to the heart. The serum transaminase levels were elevated only slightly and recovery was uneventful.

3. M.G., a 55 year-old white man with a history of myocardial infarction in January, 1958 was admitted in acute pulmonary edema on February 11, 1959. He had been on a maintenance dose of 0.25 mg. of digoxin before admission. He received 1.0 mg. of digoxin during the first 24 hours after admission and at 11 p.m. on February 12 he developed an atrial tachycardia with 1:1 and 2:1 conduction. He was given procaine amide orally every four hours and Kaon Elixir® (potassium gluconate and potassium chloride) 20 cc. stat, then 10 cc. every four hours for five doses. Digoxin was discontinued. The next day the electrocardiogram showed a normal sinus rhythm. Rales in both lung fields were still present, hence he was restarted on digoxin 0.25 mg. twice a day and was also placed on chlorothiazide 500 mg. three times a day. On February 17, after three days of chlorothiazide, atrial tachycardia with 2:1 block again appeared. Potassium therapy was reinstituted and digoxin once again was discontinued. On the same day, the rhythm reverted to normal sinus and digoxin 0.25 mg. daily was restarted two days later. He had no recurrence of atrial tachycardia, although he developed another episode of acute pulmonary edema on March 9 followed by another rise of the serum transaminase to 81, 240, and 95 units on three consecutive days. He later expired.

*Comment:* The relation of the tachycardia to digitalis is well documented in this case. The danger of lowering the potassium by the administration of chlorothiazide in a patient receiving large doses of a cardiac glycoside is evident. The same danger is present when digitalis and steroids are given simultaneously, as reported by Smagranoff and Jick.

4. H.C., a 74 year-old white woman who had had arteriosclerotic heart disease for several years and had been on digitalis intermittently, was admitted with severe right upper chest pain radiating down her right arm. Her blood pressure was 138/86 and she was in mild pulmonary edema. An electrocardiogram on October 18, 1958 showed an acute posterior myocardial infarction. Her chest x-ray film on the same day showed cardiomegaly and pulmonary congestion. On October 20, her electrocardiogram showed nodal tachycardia with a ventricular rate of 115 per minute. On October 21, she developed atrial fibrillation, and at 2:45 p.m. on the same day her right lower extremity became cyanotic and cold with neither popliteal nor dorsalis pedis pulsations. Intra-arterial injection of priscoline was tried with no success; she was too ill for any surgical intervention. On October 22, her pulmonary edema became more severe and she died on the same day.

*Comment:* Askey noted that the occurrence of supraventricular tachycardia with atrial fibrillation is extremely uncommon. Decherd and Hermann<sup>8</sup> found that such an association was 10 times more likely to occur if A-V block was present than when the latter was absent. Clinically, our case 1 also developed atrial fibrillation although no electrocardiogram was obtained.

5. W.L., a 53 year-old white man was admitted to White Cross Hospital through the emergency room at 12:40 a.m. on July 19, 1958. On the night of admission, he had

TABLE 1.—FIVE CASES OF SUPRAVENTRICULAR TACHYCARDIA REPORTED HERE COMPARED WITH 14 PREVIOUSLY REPORTED CASES

Author	Case No.	Sex	Age	Location of Infarct	Vent. Rate	Digitalis Intoxication	Remarks
Askey: <sup>2</sup> No. 1	1	M	56	?	160	No	Expired suddenly; no autopsy.
No. 2	2	F	76	anterior	210	Suspicious. SVT after 18 grams of digitalis	Expired suddenly; no autopsy.
No. 3	3	M	67	anterior	185	Yes	Expired—pulmonary edema uncontrolled.
No. 4	4	M	72	posterior	160	Yes	Autopsy showed recent posterior and old anterior myocardial infarction.
No. 5	5	M	66	anterior	230	Yes	Expired—pulmonary edema and pneumonia.
Fredermuth et al.: <sup>1</sup> No. 3	6	?	55	postero-lateral	165* with 2:1 block	No	Expired. No autopsy.
No. 6	7	?	45	posterior	150* with 3:1, 2:1 block	No	Expired. No autopsy.
No. 7	8	?	55	antero-septal	150* with Wenckebach	No	Autopsy showed acute antero-septal with rupture at the site of infarction.
No. 8	9	?	26	?	190* with Wenckebach	Yes	Expired. No autopsy.
No. 9	10	?	72	?	Simultaneous atrial and nodal tachycardia with A-V dissociation.	Yes	Expired. No autopsy.
No. 10	11	?	76	?	Atrial—150; Vent.—160	Yes	Expired. No autopsy.
Pascale et al.: <sup>3</sup> No. 2a	12	M	65	?	150	?	Expired. No autopsy.
Enselsberg et al.: <sup>4</sup> No. 1	13	M	70	anterior and posterior portions of IVS	150	Yes	Recent infarction IVS, anterior and posterior portions at autopsy.
Smagoroff et al.: <sup>5</sup>	14	F	69	lateral and diaphragmatic	Atrial—185 Nodal—110	Yes	Recovered.
Present Cases: No. 1	15	F	71	anterior and posterior portions of IVS	Atrial—156 Nodal—136	No	Recovered.
No. 2	16	M	58	posterior	188	No	Recovered.
No. 3	17	M	55	antero-septal	188* with 1:1; 2:1 block	Yes	Expired.
No. 4	18	F	74	posterior	115	No	Expired. No autopsy.
No. 5	19	M	53	anterior 7/26/58 posterior 1/4/59	273 300	No	Recovered. Acute occlusion of right anterior descending branch and old anterior infarction.

a severe, gripping, anterior chest pain of sudden onset followed by collapse. Oxygen was administered and 1 ampule of Levophed® (levarterenol bitartrate) in 500 cc. of 5 per cent glucose in water was started immediately intravenously. An electrocardiogram showed a supraventricular tachycardia with a ventricular rate of 273 per minute (Fig. 1—A, B, C). Within 15 minutes, 2800 mg. of procaine amide was given intravenously, at which time the rhythm reverted to normal sinus (Fig. 1-D) with a rate of 107 per minute. He became conscious and his color improved. At 2:30 a.m. he was admitted to his room with a blood pressure of 110/60, pulse 114, and respiration 24. Serial electrocardiograms showed the evolutionary changes of an acute antero-septal myocardial infarction. Serum transaminase determinations were 87 and 65 units and the leucocyte count was 13,550. A roentgenogram of the chest on August 6 showed a normal cardiac silhouette.

While hospitalized, he was treated with the anticoagulant Hedulin® Procain amide 100 mg. every four hours for eight doses was given intramuscularly following reversion to sinus rhythm. At no time did he receive a digitalis preparation. There was no recurrence of supraventricular tachycardia and his recovery was uneventful.

Second admission: On December 22, 1958, he was readmitted because of sudden onset of chest pain radiating to the neck. He did not complain of rapid heart action. The subsequent work-up was completely negative for acute myocardial infarction. He was discharged with the diagnosis of acute coronary insufficiency.

Third admission: On January 4, 1959, he was again admitted through the emergency room because of a sudden onset of rapid heart action followed by anterior chest pain which started only a few minutes before admission. His blood pressure was 90/60. He was conscious but pale and perspiring. His lungs were clear. The electrocardiogram (Fig. 1-E) showed a supraventricular tachycardia with a ventricular rate of 300 per minute. Carotid massage and eyeball pressure were tried with no slowing of the heart rate. Cedilanid 0.8 mg. was given intravenously and 1000 cc. of 5 per cent glucose in water with 1 ampule containing 4 cc. of levarterenol bitartrate was started intravenously. Carotid massage was repeated after 30 minutes but again with no success. A few minutes later another 0.4 mg. of Cedilanid was given intravenously. About 10 to 15 minutes after the second dose of Cedilanid, he became cyanotic and then stopped breathing. His blood pressure and pulse were unobtainable. The electrocardiogram showed ventricular fibrillation (Fig. 1-F). Artificial respiration was maintained while 500 mg. of procaine amide was given intravenously but to no avail. An autopsy showed acute coronary thrombosis of the right anterior descending branch. Serial sections along the right ventricular wall showed petechial hemorrhages in the myocardium and subendocardium, and there was an old myocardial infarction with fibrosis of the anterior and apical portions of the left ventricle.

*Comment:* This patient was apparently in good health with no known heart disease until his first attack on July 26, 1958. Between his two attacks he was free from episodes of tachycardia. He did not receive a digitalis preparation except during his third admission. The exact cause of the unexpected outcome of the last episode of tachycardia treated with Cedilanid, after the successful treatment of the first episode with procaine amide, is open to speculation. However, the fact that the heart had sustained further damage might have rendered any treatment unsuccessful.

### Discussion

The benign form of paroxysmal atrial tachycardia is familiar to everyone. Its sudden onset and cessation and its quick termination by simple measures which increase vagal tone either mechanically or pharmacologically needs no discussion here. On the other hand, the rarity and grave prognostic import of supraventricular tachycardia associated with acute myocardial infarction deserves emphasis and clarification, in the hope that this arrhythmia might be prevented with proper care of the coronary patient.

Mintz and Katz<sup>7</sup> analyzed 572 cases of recent myocardial infarction and found only three cases of supraventricular tachycardia. No details are available. Rosenbaum and Levine<sup>8</sup> reviewed the electrocardiographic features of acute myocardial infarction but found no cases of paroxysmal atrial tachycardia in 208 cases. It is interesting to note that all the patients included in their study were presumed to have had their first attack. In addition, 300 cases of acute coronary thrombosis were studied by Master et al<sup>9</sup> who found only five with supraventricular tachycardia, all of whom had heart failure, cardiac enlargement, and hypertension.

The series tabulated herein comprises a total of 19 cases including the five we are reporting. Sixteen cases proved fatal and only three recovered. Case 5 of our report recovered from his first attack but his second attack was fatal.

The relation of digitalis to the development of this arrhythmia is well documented. The possibility that digitalis intoxication may be induced by the use of steroids or chlorothiazide must be emphasized, as illustrated by Smagranoff's case and our case 3 respectively. The frequent occurrence of paroxysmal atrial tachycardia with or without block resulting from digitalis intoxication has been emphasized recently.<sup>10</sup> We have never hesitated to give patients with acute myocardial infarction digitalis when it is indicated,<sup>11</sup> but it should be stressed that this drug represents a two-edged sword, and its prudent use is required if supraventricular tachycardias are to be avoided.

At the time of the reversion of the first attack of supraventricular tachycardia to normal sinus rhythm in our case 5 with 2800 mg. of procaine amide given in 10 to 15 minutes, we were unaware of the previous use of this drug in such a massive dose given in so short a time. It should be noted that time was of primary concern in the emergency treatment of this case, hence no adherence was made to the rule of injecting the drug slowly. While hypotension results from the rapid injection of large doses of procaine amide, the hypotension that was already present in this patient was being treated with intravenous levarterenol bitartrate when the procaine amide was injected. Others have advocated the adjunct use of vasopressors in normotensives when large doses of procaine amide must be given.

The question of whether the terminal episode of ventricular fibrillation in our case 5 was induced by digitalis or by myocardial infarction is quite provoking. The successful result with procaine amide in the first attack was not obtainable with the use of Cedilanid in the second attack. The usefulness, safety, and effectiveness of Cedilanid in the treatment of supraventricular tachycardias is well substantiated. Enselberg et al<sup>4</sup> recently reviewed the literature on ventricular fibrillation due to digitalis preparations and reported 2 cases produced by acetyl strophanthidin given intravenously. The case reported by Beck et al<sup>13</sup> of ventricular fibrillation abolished by electric shock is the only report we can find that was probably due to lanatoside C. This was a 14 year-old boy who was given 1.6 mg. of lanatoside C intravenously (thought by Greene<sup>13</sup> to be an excessive dose) in the operating room for tachycardia of 160 per minute which occurred during induction of nitrous oxide and ether anesthesia.

Why is supraventricular tachycardia so infrequent in acute myocardial infarction? This problem becomes the more interesting when we contrast the frequent occurrence of benign paroxysmal atrial tachycardia in patients with no detectable heart disease. The peak incidence of benign paroxysmal atrial tachycardia falls in the 20 to 40 year age group; whereas, in our series the average age at which supraventricular tachycardia complicated myocardial infarction was 65 years. Hence, the rarity of this arrhythmia in myocardial infarction may in part represent a reflection of its decreased incidence in the older age group. Many of the factors that provoke benign atrial tachycardia may be mediated through a vagal reflex. There is no reason to think that such reflexes are not operating in a patient who sustains an acute coronary occlusion at the onset of the attack or thereafter, and yet, supraventricular tachycardia in acute myocardial infarction is rare. It is possible that the heart of the older patient is less likely to be swayed by fluctuations in vagal and sympathetic tone than that of the youth.

In the present series, digitalis intoxication and severe heart damage manifested by cardiomegaly, hypertension, heart failure and previous coronary occlusions, appear to be the common determinants in the production of supraventricular tachycardia. The altered vagal component of persons in the older age group who fall victim to coronary artery disease is probably one reason why this arrhythmia is rare in acute myocardial infarction except when complicated by severe heart damage and/or digitalis overdose.

#### SUMMARY

1. This study of 360 recently hospitalized cases of acute myocardial infarction and a review of the literature confirms the impression that supraventricular tachycardia is a relatively rare complication of acute myocardial infarction.

2. Supraventricular tachycardia is of grave prognostic import in acute myocardial infarction. This is at least partially due to the fact that this arrhythmia seems to complicate infarction mostly in patients with severely damaged hearts. Only three recoveries were noted in the 19 cases studied.

3. Of our five cases reported, one was related to digitalis intoxication and four were not. The danger of inducing supraventricular tachycardia constitutes another reason for due caution when digitalis is given to patients with acute myocardial infarction, although it should, of course, be used when indicated.

4. In one of our cases the supraventricular tachycardia converted to a fatal ventricular fibrillation following an average dose of deslanoside.

#### RESUMEN

1. Este estudio de 360 casos de infarto agudo del miocardio recién hospitalizados así como la revisión de la literatura confirma la impresión de que la taquicardia supraventricular, es una complicación relativamente rara del infarto agudo del miocardio.

2. La taquicardia supraventricular es de pronóstico grave en el infarto agudo del miocardio. Esto se debe cuando menos en parte a que esta arritmia parece complicar a los infartos en enfermos con corazón muy dañado. Sólo tres recuperaciones se observaron en los 19 casos estudiados.

3. De nuestros cinco casos relatados, uno estaba en relación con intoxicación digitalica y cuatro no. El peligro de provocar taquicardia supraventricular, constituye otra razón para la debida cautela cuando se da digital a los enfermos con infarto del miocardio aunque debe, por supuesto darse, cuando está indicada.

4. En uno de nuestros casos, la taquicardia supraventricular se convirtió en fibrilación ventricular fatal después de una dosis mediana de deslanoside.

## RESUMÉ

1. Cette étude de 360 cas récemment hospitalisés pour infarctus myocardique aigu et une revue de la littérature confirment l'impression que la tachycardie supraventriculaire est une complication relativement rare de l'infarctus myocardique aigu.

2. La tachycardie supraventriculaire est de pronostic grave quand elle est associée à l'infarctus myocardique aigu. Ceci est dû au moins partiellement, au fait que cette arythmie semble la plupart du temps compliquer l'infarctus chez les malades atteints de lésions cardiaques graves. Trois guérisons seulement furent notées dans les 19 cas étudiés.

3. Sur les cinq cas rapportés par l'auteur, l'un fut en rapport avec une intoxication par la digitale. Le danger de produire une tachycardie supraventriculaire constitue une autre raison d'avoir de solides garanties avant de donner de la digitale aux malades atteints d'infarctus myocardique aigu, encore qu'elle doive être, bien sur, utilisée quand elle est indiquée.

4. Dans l'un des cas de l'auteur, la tachycardie supraventriculaire se transforma en une fibrillation ventriculaire fatale, après une dose moyenne de deslanoside.

## ZUSAMMENFASSUNG

1. Diese Untersuchung von 360 kürzlich in stationäre Behandlung genommener Fälle von akutem Myocardinfarkt, sowie eine Literaturübersicht bestätigen den Eindruck, wonach eine supraventrikuläre Tachycardie eine relativ seltene Komplikation des akuten Myocardinfarktes darstellt.

2. Die supraventrikuläre Tachycardie ist von schwerwiegender prognostischer Bedeutung beim akuten Myocardinfarkt. Dies ist mindestens teilweise der Tatsache zuzuschreiben, daß diese Arrhythmie den Infarkt meistenteils bei Kranken mit schwer geschädigten Herzen zu komplizieren scheinen. Es wurden nur 3 Genesungen unter 19 beobachteten Fällen bemerkt.

3. Von unseren 5 mitgeteilten Fällen stand einer in Beziehung zu einer Digitalis-Intoxikation und 4 nicht. Die Gefahr der Entstehung der supraventrikulären Tachycardie ist ein anderer Grund zu einer entsprechenden Vorsicht, wenn Patienten mit akutem Myocardinfarkt Digitalis erhalten, obgleich man es natürlich benutzen muß, wenn es indiziert ist.

4. Bei einem von unseren Fällen wurde aus der supraventrikulären Tachycardie ein Kammerflimmern mit tödlichem Ausgang im Anschluß an eine mittlere Dosis von Deslanosid.

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## *SUMMARY OF CURRENT THERAPY*

### **Hypothermia in the Treatment of Cerebral Injury Occurring during Cardiac Arrest**

#### **Report of Three Cases**

Considerable interest has been expressed in the literature on the subject of cardiac arrest occurring during surgical procedures. There has been widespread dissemination of information on techniques of rapid diagnosis and therapy. Physicians in almost every large community are being offered courses in the practical aspects of this problem.

Despite all this, the dismal fact confronting us is that a great number of these cases are not being treated successfully. Many cases of circulatory arrest are being subjected to the approved techniques of thoracotomy and cardiac massage with very disappointing results. In spite of great technical advances in the surgical handling of these cases, there does not appear to be a corresponding increase in the percentage of successful resuscitations.

The major cause of failure in these cases appears to be cerebral damage resulting from cerebral ischemia. It has usually been assumed that the maximum period of circulatory arrest that can be successfully tolerated is between three to four minutes. Arrests of longer duration have been successfully resuscitated as far as restoration of cardiac action is concerned; but with death eventually occurring from the sequelae of cerebral ischemia.

The use of hypothermia to protect against ischemic damage to the brain has been extensively used clinically in performing open-heart surgery. It has long been known that hypothermia at levels of 26°-30°C. during the period of circulatory occlusion can provide safe occlusive periods of 9-20 minutes. At somewhat lower levels, safe occlusions for periods of 45 minutes and longer have been reported.

Heretofore it has been assumed that ischemic damage to the central nervous system was irreversible. In cardiac arrest patients exhibiting such damage, the salvage rate has been distressingly low. In 1958,<sup>1</sup> Williams and Spencer reported on the use of hypothermia in the treatment of eleven patients with severe central nervous system damage with seven survivors. In their report they re-confirm the rarity of survival in severely damaged patients without the use of hypothermia. In their series of cases, adequate airway and ventilation was assured by the use of tracheotomy and a mechanical respirator when necessary. Hypothermia was instituted as quickly as possible following restoration of cardiac action and the recognition of severe ischemic damage. Such damage was recognized by deep coma, fixed dilated pupils, poor or absent respiratory movements, and areflexia. Cooling was maintained at levels of 31°-33°C. Cooling and the use of the respirator were continued for periods of time up to 96 hours until the patients showed evidence of return of cerebral function or died. Patients who developed metabolic acidosis because of the inadequate circulation or ventilation were given bicarbonate intravenously.

The exact mechanism by which reversibility of the cerebral damage is achieved is not completely understood. It must be assumed that the actual death of nervous tissue does not occur immediately, but occurs over continuing periods of time possibly as a result of severe cerebral swelling caused by the ischemic injury.<sup>14</sup> In this brief report we do not have the space to go into the neurological literature on this subject. However, we have included many of the reviewed articles in the bibliography.

We have had occasion to use hypothermia in three cases of cardiac arrest with severe central nervous system damage. All three were treated with the same basic method. Of these, there were two complete neurological recoveries and one death. The non-surviving patient died on the fifth day with no evidence of return of cerebral function. One of the surviving patients was a young woman who suffered an arrest during delivery. As soon as the mother's cardiac action was restored, the infant was hurriedly delivered. The infant's delivery was completed before any drop in the mother's temperature occurred. As far as it can be ascertained at the present time, the infant does not have any neurological damage. However, it may be some time before an accurate assessment can be made. The infant, although quite cyanotic when delivered, did breathe and cry adequately shortly afterwards and was not treated with hypothermia.

In order to demonstrate the method of handling, we will describe the other survivor in some detail.

The patient was a female of 42 years of age with no previous record of serious illness of any type. She was undergoing varicose vein ligation and stripping, and was given a spinal anesthetic supplemented by an intravenous barbiturate for analgesia. As is usual in these cases it was quite difficult to ascertain the total time elapsed before effective cardiac action was restored. In addition, it was impossible to determine the period of severe anoxia that preceded the arrest. It was our impression that a period of arrest of at least 5-6 minutes was experienced. Following resuscitation, the patient was deeply comatose, the pupils were widely dilated and fixed, the respiratory movements were quite shallow and would lapse into a Cheyne-Stokes pattern. It was necessary to maintain an adequate blood pressure with vasopressor agents for a period of time following restoration of cardiac action. While the chest was being closed, large plastic pillow-cases filled with ice were placed under and about the patient. At no time was shivering noted. Pressure points on the skin were coated with a lanolin ointment as a protection against frost-bite. When the chest closure was completed it was noted that the respiratory movements were too slow and shallow to sustain life. Accordingly, a tracheotomy was performed and the patient was connected to a Mörch respirator set at a rate of 18 per minute with a tidal volume of about 600-800 cubic centimeters. Within 2 minutes all evidence of voluntary respiratory efforts ceased and the respirator maintained the desired rate and tidal volume with no interference from the patient. The temperature reached a level between 32°-34°C within one hour and was maintained there.

After 8 hours the patient developed severe irritability with wild uncoordinated jerking of the extremities and muscular twitchings upon even

very slight stimulation. At 12 hours coordinated motions in response to stimuli appeared, and when the respirator was discontinued, adequate deep regular respiratory movements commenced. At 18 hours the patient responded to stimuli by opening the eyes, and the hypothermia was discontinued. However, it was necessary to use the ice bags to keep the temperature at normothermic levels as the temperature tended to rise to hyperthermic levels quite rapidly. By 24 hours the patient would open her eyes and make attempts to speak but appeared extremely depressed and drowsy. This persisted another 24 hours at which time she made what appeared to be a complete neurological recovery. Her only residual neurological defects were a complete loss of memory of the preceeding two weeks and bilaterally positive Babinski's which persisted for several weeks.

### Discussion

The maximum improvement in survival that can be expected from intensive education in recognition and surgical treatment after cardiac arrest has been achieved. However, we are now seeing cases in which it has been possible to restore cardiac action, but in which the severe cerebral injury results in death. In spite of, or possibly because of our education and training in promptly instituting surgical treatment in all cases of cardiac arrest, we can expect to have patients in whom the resuscitation required more than the critical 3 or 4 minutes.

If we are to further improve our statistics it is imperative that we recognize the fact that cerebral injury resulting from ischemia may not be irreversible. Prompt cardiac resuscitation should be followed by prompt institution of hypothermia. Adequacy of ventilation should be assured by the use of a mechanical ventilator and tracheotomy when indicated. The metabolic acidosis resulting from inadequate respiration and circulation can at least be partially remedied by the use of bicarbonate intravenously. By using these techniques energetically even in supposedly hopeless cases, a further improvement in survival statistics can be achieved.

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## ELECTROCARDIOGRAM OF THE MONTH

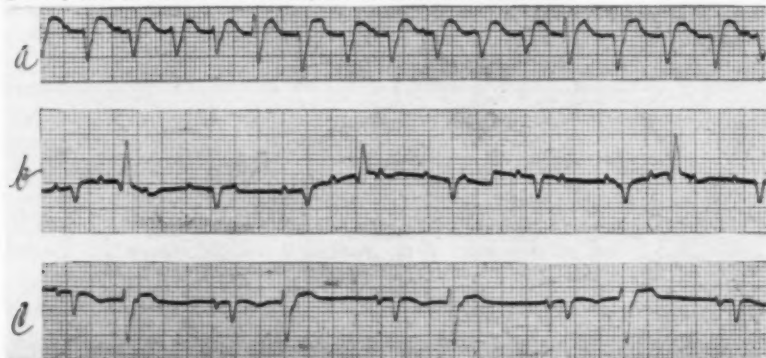
### Various Arrhythmias Due to Digitalis Intoxication Recorded in the Same Patient in the Course of One Month

From a woman of 64 with congestive heart failure. All tracings lead V-1, taken on different days.

a: Recorded when she had been on Digoxin for some time, amount and length of period not ascertainable; medication stopped that day. Ectopic ventricular tachycardia of about 158, independent atrial tachycardia of about 150. The slight difference in rate resulted in a slow shift between the two rhythms, P waves gradually approaching the QRS complexes (see in particular the first and last two beats) and then becoming hidden in them and the T waves. When an atrial impulse occurred sufficiently late after the ventricular one to reach the junctional tissues outside their refractory period, it was conducted to the ventricles, resulting in a premature ventricular response having a different shape (the fifth and twelfth beats): dissociation with interference.

b: Recorded three weeks after (a). In the interval she had received Digoxin intermittently, but none the last two days prior to this record. Atrial tachycardia of 150-166 and independent A-V nodal tachycardia of 79-81 with, either some supraventricular beats conducted to the ventricles with aberrant ventricular conduction (dissociation with interference), or ventricular extrasystoles. Note that the interval following the differently shaped ventricular complexes equals the cycle length of the automatic A-V nodal rhythm, that is, these beats resulted in a shift of the A-V nodal rhythm.

c: Recorded five days after (b), during which she had not received digitalis. Bigeminy due to ventricular extrasystoles; sinus rate about 88.



Seventeen days after the record (c) was obtained, she died of an anterior myocardial infarct. A cardiogram recorded on the day before death showed sinus tachycardia of 110-115.

The reproduced tracings demonstrate various arrhythmias known to occur as a result of digitalis intoxication. Of these, ectopic ventricular tachycardia has been recognized for a long time, whereas the precipitation of atrial tachycardia by this drug has only been appreciated more recently. Regarding (b), A-V nodal tachycardia of this comparatively low rate, often with A-V dissociation, has been reported as an arrhythmia *sui generis* and being due to digitalis in a proportion of cases. Both possibilities considered as the mechanism underlying the differently shaped ventricular complexes would account for the slight variations in their shape, and also for the time relations: in the event of their being aberrantly conducted supraventricular beats in dissociation with interference, the shift of the A-V nodal rhythm would be due to their depolarising the A-V centre on their way; in the event of ventricular extrasystoles the condition would resemble that prevailing in complete A-V block with ventricular extrasystoles. After digitalis was withheld for one week, a marked drug effect was still present as revealed by the record (c).

## X-RAY FILM OF THE MONTH

### *Clinical Information*

M.L., a woman of 29 years, delivered her second child in September, 1958. Her menstrual periods became irregular subsequent to January, 1959. On May 15, 1959, she complained of sudden onset of dyspnea associated with pleuritic pain in her left lower chest. Her temperature was 102°F. The following day all symptoms had disappeared but her roentgenogram showed a blurred outline to the left hemidiaphragm, which was elevated 1-2 cm. A small collection of pleural fluid was present in the lateral costophrenic recess (Fig. 1).

On admission to the hospital, at the end of May, she was symptomless and looked well. There was diminished movement, dullness to percussion, decreased vocal resonance and decreased breath sounds over the left lower chest. The heart sounds were normal. The roentgenogram on June 2, 1959 (Fig. 2), showed remarkable enlargement of the cardiac outline to the left and apparent elevation of the left hemidiaphragm which was now smooth and convex upward. A thin line of pleural fluid was still present above the left costophrenic recess, the angle of which was acute. There was no gas shadow in the stomach. On fluoroscopy the "left hemidiaphragm" moved well. The electrocardiogram was normal; the sedimentation rate was 68 mm. in one hour. The tuberculin test was positive.

A roentgenogram in the supine position, made immediately after viewing that shown in Fig. 2, showed restoration of the cardiac outline to normal and a large effusion evenly distributed over the left lung (Fig. 3). Aspiration revealed a cloudy fluid with red blood cells, lymphocytes and a protein content of 5.8 Gm. per cent. Smear and cultures were negative for tubercle bacilli. Uterine curettage showed typical endometrial tuberculosis. She was treated with streptomycin, isoniazid and P.A.S. and the effusion had completely cleared in seven weeks (Fig. 4). Her menstrual periods have returned to normal.



FIGURE 1

FIGURE 2

FIGURE 1: P. A. film (May 16, 1959) showing small effusion at left base.

FIGURE 2: P. A. film (June 2, 1959) showing marked elevation of left "pseudo-diaphragm" with abrupt downward inclination peripherally with apparent cardiac enlargement. Note almost total absence of fluid over lateral costal surface.



FIGURE 3

FIGURE 4

FIGURE 3: A. P. 6' supine film (June 2, 1959) showing almost complete absence of mediastinal and sub-pleural fluid which has now collected along the lateral costal surface due to change in gravitational force. FIGURE 4: P. A. film (July 27, 1959) post treatment normal film.

The presence of subpulmonary effusion was suggested by the gradual upward inclination of the elevated pseudo-diaphragmatic contour with sharp drop near the costal margin, smooth outline with normal mobility, and a minute collection of fluid in the lateral gutter. The apparent cardiac enlargement was thought to be due to pericardial effusion but proved to be an intrapleural collection of fluid along the mediastinal surface. Normally, the gas shadow in the stomach lies 1 cm. below the diaphragm and if this distance is greater it may be the key to recognition of subpulmonary effusion. In this case downward displacement of the gastric air bubble was not observed, as the fundus was devoid of gas.

### Conclusion

Establishing the presence of the paramediastinal fluid was considered important as it precluded possible pericardial tap or angiocardiography. Paramediastinal effusion is rare and the mechanism involved is not fully understood. The localization of fluid below the lung is due to gravity. Medially it may be the result of partial collapse of the medial portion of the lower left lung. The pleural space was free of adhesions as shown by the immediate redistribution of the fluid with change in gravitational force when the patient assumed a supine position.

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## Case Report Section

### Atrial Flutter with 1:1 A-V Conduction\*

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To date, there have been fewer than 50 cases of atrial flutter with 1:1 A-V conduction documented in the English literature.<sup>1</sup> The purpose of this paper is to emphasize the diagnostic facets of this relatively uncommon rapid ectopic rhythm and to review the pharmacology of the various therapeutic agents used in its treatment. Two additional cases of 1:1 atrial flutter are reported, one occurring in a patient with chronic constrictive pericarditis.

#### Case Reports

**Case 1:** This 59 year-old white man was admitted to the Veterans Administration Hospital, Coral Gables, in 1957 because of chills, fever, sweats, cough and dyspnea. There was a past history of bronchiectasis, obstructive emphysema and pulmonary infarction.

The patient appeared markedly cyanotic and dyspneic. His blood pressure was 120/80, and the apical pulse was 140 and regular. There was marked clubbing and cyanosis. Severe wheezing associated with rales and diminished breath sounds was heard throughout both lung fields. The edge of the liver was 3 cm. below the right costal margin, and tender. The laboratory work-up revealed respiratory acidosis, with hypoxia and CO<sub>2</sub> retention. An electrocardiogram was interpreted as showing atrial flutter with 2:1 A-V block and cor pulmonale (Fig. 1). The atrial rate was 280/minute, the ventricular rate 140/minute. Prior to death the patient had frequent episodes of 1:1 A-V conduction, unresponsive to carotid sinus pressure (Fig. 2). Intravenous acetyl strophanthidin given under electrocardiographic control, however, increased the degree of A-V block. In spite of all measures, the patient's course was progressively downhill. Autopsy disclosed bronchiectasis, severe pulmonary emphysema and cor pulmonale.

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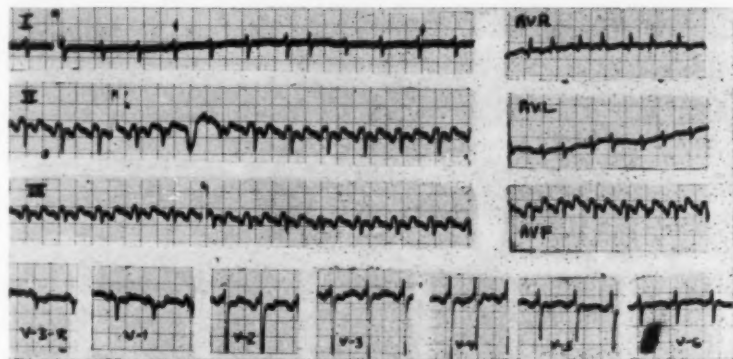
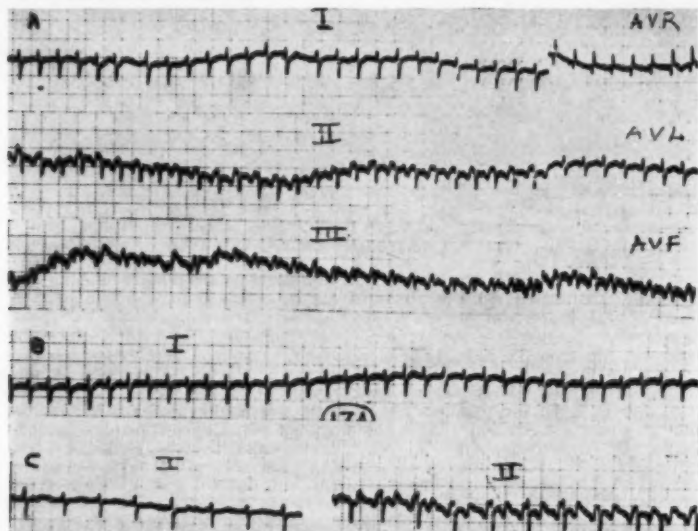


FIGURE 1—Case 1: Control electrocardiogram before onset of 1:1 A-V conduction. Atrial rate, 280; ventricular rate, 140. There is atrial flutter with 2:1 A-V conduction (occasionally 3:1 conduction). Incomplete right bundle branch block and a rare premature ventricular contraction are also present.

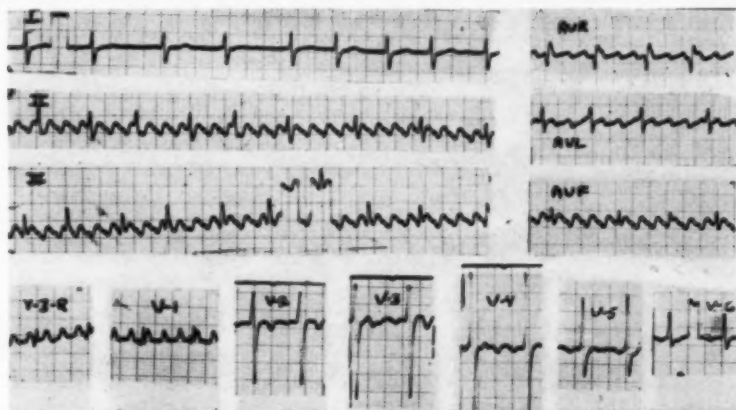
**Case 2:** A 24 year-old white man was admitted to the Veterans Administration Hospital, Coral Gables, in 1956 for episodes of palpitation, paroxysmal dyspnea and weakness precipitated by exertion or excitement. Past medical history was negative. Physical examination revealed a regular pulse of 140 per minute with a blood pressure of 110/90. There were signs of minimal right-sided heart failure. A chest x-ray film suggested calcification of the pericardium. An electrocardiogram revealed atrial flutter with varying degrees of A-V block and right ventricular hypertrophy. Cardiac catheterization was consistent with a diagnosis of constrictive pericarditis. Preoperative digitalis and quinidine failed to convert the atrial flutter to normal sinus rhythm. At thoracotomy, a thickened, partially calcified pericardium was excised and his post-operative course was uneventful.



**FIGURE 2—Case 1:** A. Six standard lead electrocardiogram showing atrial flutter with 1:1 A-V conduction. In lead I there is one brief period of 2:1 conduction. Atrial and ventricular rates are 300 per minute.

B. Long leads I and II showing continuous atrial flutter with 1:1 A-V conduction. The rate is 300 per minute.

C. Standard leads I and II after intravenous acetyl strophanthidin showing atrial flutter with 2:1 block: Atrial rate 300, ventricular rate 150 per minute.



**FIGURE 3—Case 2:** Control electrocardiogram demonstrating atrial flutter with varying degrees of block. Atrial rate 300. There is evidence of incomplete right bundle branch block with probable right ventricular hypertrophy.

Shortly after discharge, he was readmitted for severe palpitation brought on by exertion. A control electrocardiogram showed atrial flutter with 2:1 block; the atrial rate was 300/minute (Fig. 3). Minimal exertion precipitated an episode of 1:1 A-V conduction (Fig. 4). He experienced dyspnea, weakness, and severe palpitation coincident with his rapid heart action.

With re-digitalization, a high degree of A-V block was obtained and the patient has remained relatively asymptomatic. Several trials with large doses of quinidine by mouth failed to produce a normal sinus rhythm.

### Discussion

Atrial flutter with varying degrees of A-V block is not uncommon. Katz and Pick<sup>2</sup> observed an incidence of 0.54 per cent in patients having electrocardiograms at the Cook County Hospital.

Certain well-defined criteria have been considered necessary to establish a diagnosis of atrial flutter.<sup>2</sup> The atrial rate should range between 225-360 per minute with constant atrial activity, manifested by the absence of an iso-electric period measuring 0.04 second or more. Often a sharp upstroke of the flutter wave and gradual downstroke is observed which is manifest electrocardiographically as the typical "saw tooth" appearance of the flutter waves. Flutter waves are usually perfectly regular and are best seen in standard leads I and II. There is occasionally some lengthening of the F-R interval and this accounts for the slight variation in the R-R interval which may be observed. Due to the usual presence of A-V nodal block of varying degree, the ventricular rate in atrial flutter usually ranges from 75 to 160 per minute.

To aid in determining whether a rapid ectopic rhythm is atrial flutter with 1:1 A-V conduction, the presence of atrial flutter with higher grades of block either before or after the onset of the 1:1 conduction is often necessary. The occurrence of auricular fibrillation or atrial flutter with a high grade of A-V heart block following digitalis therapy has also been observed to be helpful in defining a rapid ectopic rhythm as atrial flutter with 1:1 conduction.

When 1:1 A-V conduction has been observed, symptoms have included dyspnea, palpitation, syncope, angina pectoris and heart failure.<sup>2</sup> It has been shown that with ventricular rates above 180 per minute, there is a precipitous fall in both cardiac output and coronary blood flow.<sup>4,5</sup> This is not an absolute figure and an individual patient may have relative coronary insufficiency and myocardial ischemia at a slower ventricular rate.

While atrial flutter with varying degrees of A-V block is usually diagnosed readily with the aid of the electrocardiogram, 1:1 A-V conduction has been confused with paroxysmal atrial, nodal and ventricular tachycardias and ventricular flutter. The ventricular complexes may be widened as a result of organic disease or as a result of the so-called "fatigue" of a bundle and add to the difficulty in establishing a correct diagnosis. Various attempts to increase vagal tone may be of invaluable diagnostic aid by increasing the degree of A-V block, and thereby providing an opportunity for clarification of the atrial mechanism.<sup>6,7</sup>

Digitalis, quinidine, and procaine amide have been recommended for the treatment of atrial flutter.<sup>2,6,8,9,10-15</sup> Digitalis is generally considered the drug of choice in initiating the therapy of rapid atrial flutter.<sup>1,2,6,8-13</sup> Both by a direct effect on the A-V node

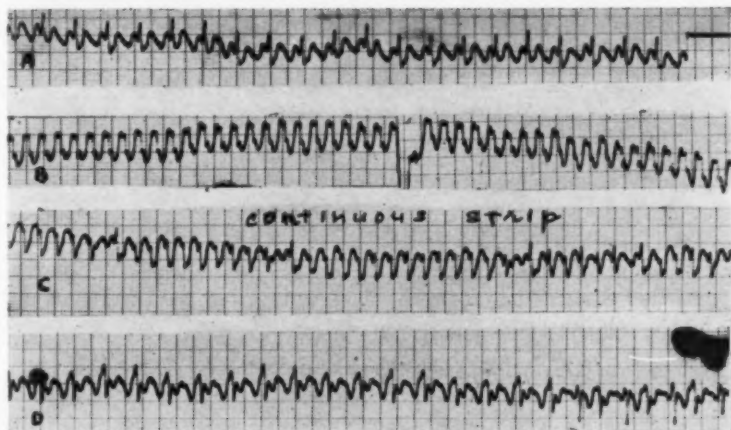


FIGURE 4—Case 2: A-D: Lead II taken during paroxysm of severe palpitation showing alternation from atrial flutter with 2:1 A-V conduction to 1:1 A-V conduction and back to 2:1. Atrial rate 310. C and D are a continuous strip.

and by a vagotonic action, digitalis slows A-V nodal conduction and protects the ventricle from the rapidly discharging atria.

Quinidine and procaine amide may terminate atrial flutter or atrial fibrillation by conversion to normal sinus rhythm. This appears to be accomplished by both direct and vagolytic effects on the atria. According to the classical concept of Sir Thomas Lewis,<sup>14</sup> if the refractory period is increased to a greater degree than the conduction is slowed, then the head of a circus wave confronts refractory muscle and is stopped.

More recently the circus movement theory has been challenged<sup>15</sup> and the concept of a rapidly discharging ectopic focus presented as an explanation for the atrial arrhythmias. Accordingly, the beneficial effects of quinidine and procaine amide in atrial flutter or fibrillation can be explained by their depressant effect on rapid ectopic auricular pacemakers.

Quinidine and procaine amide have often been considered etiological factors in the production of 1:1 A-V nodal conduction in patients with established atrial flutter.<sup>6,8,9,12,13,16</sup> This dangerous complication of quinidine and procaine amide therapy results from a transient vagolytic effect on the A-V node. This increase in A-V nodal conduction, associated with drug induced slowing of the atrial flutter rate, may convert an atrial flutter rate of 340 per minute with 2:1 A-V nodal conduction to an atrial flutter with a rate of 280 to 300 per minute with 1:1 A-V conduction.

It would appear, therefore, that any attempt to convert atrial flutter to normal sinus rhythm with quinidine or procaine amide is hazardous if the conduction through the A-V node has not been previously delayed by adequate digitalis therapy.

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## Mesothelioma Developing in the Wall of a Pulmonary Cyst

JOHN STORER, M.D., F.C.C.P. and ROBERT HOOPER, M.D.\*  
Cleveland, Ohio

Insofar as we have been able to ascertain, there is no reported case of a mesothelioma developing in the wall of a pulmonary cyst. The following is a report of such a lesion:

D.F., a 47 year-old white man, was admitted to Huron Road Hospital on March 21, 1958. He had not felt completely well for the past year, complaining of lethargy and easy fatigability. Three months prior to admission, he developed pain in both knees and ankles and was treated for arthritis. About three weeks prior to admission he noticed clubbing of the fingers, periodic elevation in temperature, accentuation of his lethargy and the onset of a cough productive of mucoid sputum flecked with blood on occasions. In the two weeks prior to admission, he lost 7 pounds. On clinical examination he appeared to be acutely ill. His temperature was 38°C. by mouth, pulse rate 100 and respirations 25. There was marked clubbing of the fingers and toes, but no visible cyanosis and no other cutaneous lesion. The pertinent findings were confined to the respiratory system. There were coarse rales audible over the right upper posterior one-third of the chest. There was no other abnormal finding.

The patient had been subjected to a chest x-ray film inspection yearly at his place of employment. A film dating to 1955 showed a cystic lesion in the upper lobe of the right lung (Fig. 1) which had not changed on three repeat yearly examinations. A chest x-ray film taken at the time of admission (Fig. 2) disclosed the presence of a thin-walled cavity in the anterior inferior portion of the upper lobe of the right lung. The inferior wall of the cavity which measured about 3.5 cm. in diameter was quite thickened and appeared to be umbilicated. This projected into the lumen of the cystic lesion. This, as compared with the previous films, represented a new finding. Scattered through the remaining portion of the upper lobe of the right lung was an infiltrative lesion appearing inflammatory. X-ray film examination of the forearms and legs showed no evidence of periosteal thickening. A tuberculin test with first strength PPD was positive. Hemoglobin was 85.2 per cent, white blood cell count 11,600, 69 Polys., 29 Lymph. and 2 Monocytes. Urinalysis was not remarkable. Serology negative. Sputum examination revealed the presence of beta hemolytic streptococcus.

An electrocardiogram showed a tendency toward right axis deviation, but otherwise was not remarkable. Pulmonary function studies disclosed a maximum breathing

\*From the Department of Thoracic and Cardiovascular Surgery, Huron Road Hospital.



FIGURE 1

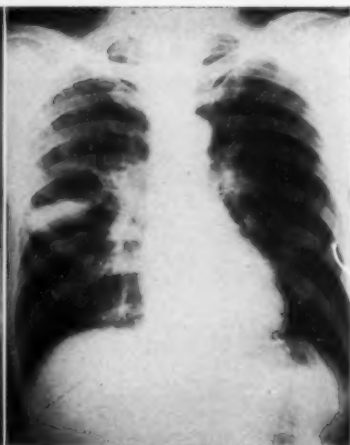


FIGURE 2

FIGURE 1: Chest film (July, 1955) disclosing cystic lesion in right upper lobe.

FIGURE 2: Marked change in the cyst noted in March, 1958.

capacity of 124 liters (135 liters predicted normal), walking ventilation 11.00 L/M<sub>2</sub>/M, Surgical Index .10 and vital capacity 95 per cent of normal with an expiration time of 6.5 seconds. Fifty-seven per cent of the total was expired in the first second. On bronchoscopy some inflammatory exudate was visualized in the lumen of the right upper lobe bronchus, but no other abnormality was noted. The aspirated secretions for cytologic evaluation were negative.

He was subjected to surgery seven days following admission. A standard right thoracotomy was done and in the right upper lobe near the minor fissure there was a cystic lesion measuring approximately 3 cm. in greatest diameter (Fig. 3). This was thought to represent the lesion visualized on x-ray film and since the middle and lower lobes were not remarkable on palpation right upper lobectomy was carried out without incident. His post-operative course was complicated by the development of a partial pneumothorax about 8 days following surgery. This necessitated the reinsertion of a thoracotomy tube in the second anterior intercostal space. This was allowed to remain in place for three or four days, following which time the lung completely expanded and the pleural space was obliterated. The specimen was submitted to the Department of Pathology and a diagnosis of malignant mesothelioma developing in the wall of a pulmonary cyst was returned. He was discharged on April 23, 1958 in good condition. Following discharge, he developed a hacking cough productive of whitish mucoid secretion occasionally tinged with blood. He was subjected to x-ray therapy on an ambulatory basis. An anterior portal dose of 2500 Roentgens and a posterior portal dose of 200 Roentgens were delivered over several days. His condition gradually deteriorated and it was necessary to re-admit him on May 22, 1958. A small space had developed and in order to obliterate it, thoracoplasty was



FIGURE 3: Operative specimen showing tumor in the cystic lesion.

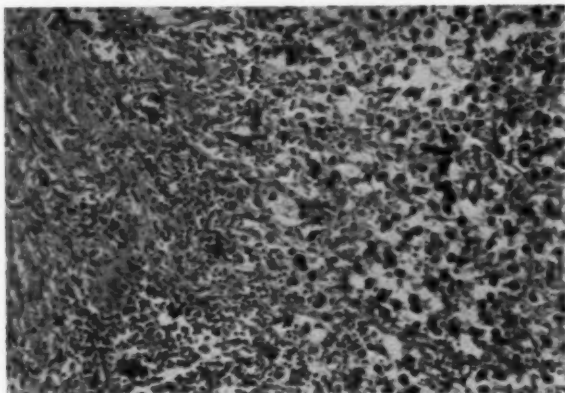


FIGURE 4: Wall of cyst far left. (high power)

done. He gradually improved following this procedure and had diminution in all symptomatology and was discharged on June 6, 1958. However his debility soon reappeared along with his elevation of temperature and a severe hacking cough. He was re-admitted on June 29, 1958 with evidence of a bronchopleural fistula and in rather severe respiratory distress. Despite the conventional measures which were carried out in a vigorous fashion, he ran an inexorable downhill course and expired on September 7, 1958. It is presumed that he died from generalized metastasis from his original lesion. Unfortunately, permission for post-mortem examination was not obtained.

### Discussion

It seems apparent that the pulmonary cyst antedated the onset of a malignant tumor in its wall. The pulmonary cyst was visualized on an x-ray film at least three years before the development of symptomatology.

The therapeutic implication in the treatment of a pulmonary cyst is obvious. Even though the occurrence of a malignant tumor in the wall of a cyst is rare, it must be considered a definite possibility. It is our feeling, therefore, that not only because of this possibility, but the other complications of bronchogenic cyst, such as suppuration, presents sufficient indication to warrant resection upon the diagnosis. No such clear cut indication exists for cysts of alveolar origin however. The time honored criteria for surgical intervention in these lesions has been the onset of pulmonary ventilatory insufficiency because of the large size. However, the differential diagnosis of bronchogenic and alveolar cysts sometimes is a matter of delicate clinical judgment. It is our opinion that an aggressive surgical approach is preferable to a policy of watchful waiting. Certainly no service is done to a patient when one has watched and waited for a malignant tumor to develop. The golden opportunity for cure represented by that most ideal form of medical therapy, i.e.: prophylaxis, has been lost when such a situation transpires.

## Schistosomal Bronchiectasis\*

K. I. JAWAHRI, M.D., and A. SHAMMA, M.D.

Baghdad, Iraq

Schistosomiasis in Iraq is an ancient disease. Ova and the adult worms of *S. hematobium* species are found in the bladder, rectum, lungs and other organs of the body with prolonged chronic irritation, granuloma formation and fibrosis. Among 2276 autopsies<sup>1</sup> 113 cases (5 per cent) had *S. hematobium* involvement of the urinary bladder. In the period 1956-1958, 39 of 270 autopsied cases had urinary schistosomiasis. Ten of these

\*From Central Pathology Institute.



FIGURE 1: Gross lung specimen, with irregular dilatation and thickening of the bronchi, typical of bronchiectasis.

had pulmonary involvement, with granulomas found in the following sites: arterial intima and in the lumen, arterial adventitia, periarterial tissue and pulmonary alveolar septa, leading to obliterative arteriolitis.<sup>1</sup> Only one case of bronchiectasis was discovered, in which the bronchial walls were involved by the ova and the case is discussed in this paper.

Among other important factors in the etiology of bronchiectasis are congenital weakness of the bronchiolar musculature with hyperdistention, obstruction to a main bronchus or group of bronchioles either by foreign body, adenoma, enlarged caseous lymph node, malignant nodules, inflamed material or exudate, bronchopneumonia in childhood, atelectasis of the related parenchyma and bronchioles with infection, excessive pull of parenchyma in atelectasis and other factors that lower the resistance of bronchial walls.<sup>3,4</sup>

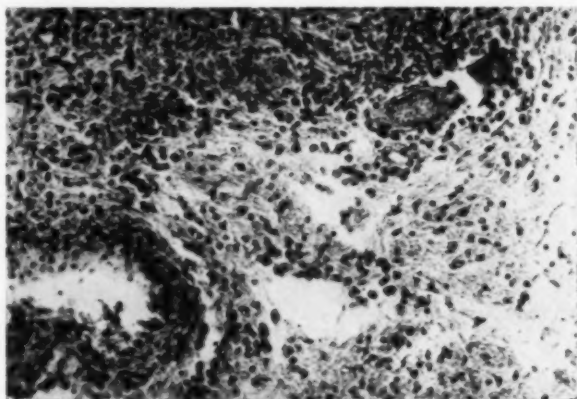


FIGURE 2: Microscopic focus of inflammatory reaction from the lung, showing an ovum of schistosoma in the lung. X150

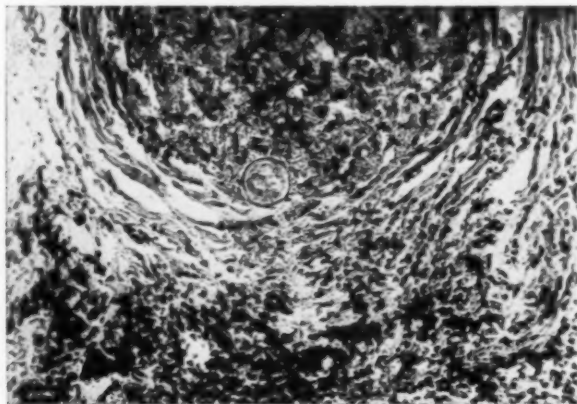


FIGURE 3: Fibrotic inflammatory nodule from the same lung, showing an ovum of schistosoma cut transversely. X150

**Case Report:** A 27 year-old married woman complained of a severe cough, copious amount of expectoration and hemoptysis. The condition started three and a half years ago with sudden rigor, and fever then developed with the above-mentioned symptoms. X-ray film and sputum examinations were made in a tuberculosis dispensary, the case diagnosed as pulmonary tuberculosis, treated, and the condition improved. Later, she was admitted to the Twaitha Hospital Tuberculosis Sanatorium for further investigation. Sputum both examined directly and by culture was always negative for tuberculosis. X-ray film disclosed pulmonary fibrosis with small areas of calcification and bronchiectasis. Urine examination revealed ova of *S. hematobium*. At last lobectomy was decided upon, and the specimen was sent to our institute for histopathological examination.

**Macroscopical Examination:** The specimen received was a dissected lower lobe of lung in which the bronchi were thickened and stiff. Their mucosa was thick, hyperemic, soft, rugose, and ulcerated. Bronchi were filled with muco-purulent exudate. The parenchyma immediately surrounding the dilated bronchi was partly or completely atelectatic and fibrotic, and the surface gave a grating sensation due to presence of calcification and the thickened bronchi. The hilar lymph nodes were enlarged.

**Microscopical Examination:** There were numerous ova of *Schistosoma hematobium* distributed throughout the lung tissue, including the bronchial walls, blood vessel walls, and alveolar septa. The majority of these ova were calcified. The blood vessels showed obliterative endarteritis, with markedly thickened walls and narrowed lumens. Rare foci of healed tuberculosis were seen. The alveolar walls were markedly thickened with small focal areas of fibrosis present. There were pus cells and other acute and chronic inflammatory cells, particularly around dilated and necrotised bronchi and bronchioles. Emphysematous changes and heart failure cells were seen. The hilar lymph nodes were heavily infected by ova of *Schistosoma hematobium* and had anthracosis.

### Comment

Pulmonary schistosomiasis is not uncommon, but as an etiological factor in bronchiectasis it is not mentioned in literature. We attribute the etiology of our case to schistosomiasis on the following basis:

- (1) hilar lymphatic enlargement develops, due to schistosomal infestation,<sup>3</sup>
- (2) bronchial wall bilharziomas (*Schistosoma granulomas*) are formed,<sup>1</sup>
- (3) marked obliterative endarteritis lowers the resistance of the bronchial walls,<sup>4</sup>
- (4) scars of granulomas formed from loose and dense connective tissues with calcified and non-calcified ova are found also in the parenchymatous lung tissue.

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# Recommendations of the Committee on Nonsurgical and Drug Therapy

COUNCIL ON PULMONARY RESEARCH  
AMERICAN COLLEGE OF CHEST PHYSICIANS

The Committee on Nonsurgical and Drug Therapy of the Council on Pulmonary Research reviewed the following subjects with these recommendations:

## *Nonsurgical Treatment of Malignant Disease of the Lungs*

1. Radiation therapy is the most effective form of nonsurgical treatment available at the present time.
2. Nitrogen mustard, which is the most widely used of the chemotherapeutic agents in the treatment of malignant disease of the lung, has but a limited palliative effect upon the cancer, no matter how it is administered.
3. Radioactive gold and nitrogen mustard both appear to have some beneficial effect upon malignant pleural effusions.
4. Hormones may on occasion afford some palliative benefit in malignant disease of the lung. The effect is not specific and is highly unpredictable.
5. Many new chemotherapeutic agents—antibiotics, antimetabolites, and alkylating agents—are being investigated in the study of treatment of malignant disease, and it is to be hoped that such studies will eventually prove fruitful.

## *The Use of Corticosteroids in the Treatment of Pulmonary Tuberculosis*

1. Benefit can be expected from the use of corticosteroids in the treatment of the acute forms of miliary, meningeal and pneumonic tuberculosis.
2. They are of value in the treatment of pulmonary tuberculosis associated with marked toxicity.
3. Their use is mandatory in patients with tuberculosis in whom there is evidence of adrenal insufficiency.
4. Corticosteroids are of value in the treatment of patients who are allergic to the use of antituberculosis drugs.
5. Patients with tuberculosis who are also afflicted with another serious disease, such as one of the collagen diseases, in which corticosteroids are indicated, should be continued on this form of therapy.
6. Corticosteroids should not be administered to patients with tuberculosis unless clinical and bacteriological sensitivity to the major antituberculosis drugs has been demonstrated and these drugs are being concomitantly administered.
7. The amount of steroid to be administered must depend upon the individual and the nature of the disease to be treated.
8. Maintenance therapy, when indicated, should be reduced to the lowest effective level as soon as permissible.

9. It is the Committee's opinion that patients with sarcoidosis should receive antituberculosis drug therapy at the same time that corticosteroids are employed.

10. Patients who have been on appreciable amounts of corticosteroids within a two-year period should receive the drug both preoperatively and postoperatively if major surgery is to be undertaken.

*Home Care of the Geriatric Patient with Tuberculosis*

The Committee is of the firm opinion that the geriatric patient with tuberculosis should be hospitalized for treatment whenever possible. If this is not possible, therapy should at least be initiated in the hospital. In those instances in which the patient will not accept such advice, the Committee recommends that the following principles be followed:

1. The minimal goal of therapy is the reversal of infectiousness.
2. The family and the community must be protected. Children should not be allowed in the home.
3. Close medical supervision must be maintained.
4. Treatment with INH and PAS is recommended. Older patients may object to the use of PAS. In such cases the combination of INH and streptomycin should be used.
5. Treatment should be continued for a minimum period of 24 months, providing the disease process has become inactive in that time. If evidence of activity persists, chemotherapy should be employed for the remainder of the patient's life.

COMMITTEE ON NONSURGICAL AND DRUG THERAPY

Council on Pulmonary Research  
American College of Chest Physicians

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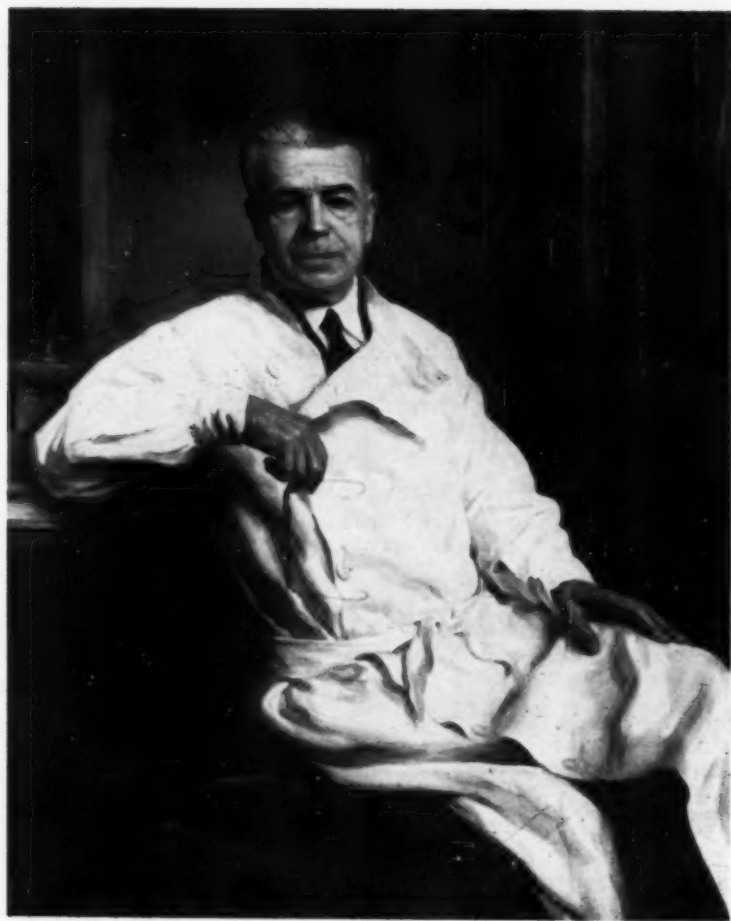
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**Professor Lopo de Carvalho**

## PRESENTATION OF THE INTERNATIONAL COLLEGE MEDAL\*

ANDREW L. BANYAI, M.D., F.C.C.P.\*\*

Chicago, Illinois

Ladies and Gentlemen: My assignment is to present to you the man who is about to receive the international medal of the American College of Chest Physicians for meritorious scientific achievement.

Should I just enumerate his accomplishments, it would be a mere protocol. Should I only praise his high attainments, it would be a mere eulogy. Instead, I shall offer you outlines of a sketch which may portray a true picture of a great man and an outstanding physician.

To be sure, his lifetime devotion has been to the welfare of his patients. His incessant dedication to medical education has been for the benefit of disciples the world over. His indefatigable research work has been in quest for better means of diagnosis and treatment.

Ever since the early days of his professional career, his interest followed the path the milestones of which bear the names of pioneers in clinical and basic sciences. One of these is that of Wilhelm Konrad Roentgen whose genius, not too far from here at the University of Wuerzburg, Germany, discovered the x-ray 65 years ago. In our own field of specialty, this great discovery made it possible to photograph in the living the heart and the lung and to inspect them fluoroscopically.

Further developments along these lines brought about the use of contrast medium for the roentgenologic visualization of the bronchial tubes. Even so, in spite of clear-cut x-ray delineation of the lower air passages in this manner, pulmonary markings have remained a puzzling riddle. Because of this, a great many diagnoses of chest diseases got caught and entangled in the seemingly inscrutable network of x-ray pattern of the lung. To many of us who have been doing what is candidly called interpretation of roentgenograms of the chest, often times the so-called normal markings of the lung posed like secret telegraphic codes or like undeciphered hieroglyphs—until systematic and purposeful research work began to unravel their substrata.

The man who started this work in 1930 has done away with theory and assumption when he made these lung shadows reveal their identity, when he converted a maze of lines into meaningful ideograms, when, as if by the touch of a magic wand, he changed obscure images into intelligible semeiology of daily practice of medicine. Through his uncanny inquisitiveness and flawless methodology, and with relentless perseverance, he succeeded in devising a new, serviceable, superior method for superior diagnosis.

In this manner, it became possible not only to clearly outline these markings in roentgenograms, but also to identify them as the vascular structures of the lung. Also, it became possible to determine the competence of blood flow in the pulmonary arteries, veins and even in the capillaries. The very same method is being used for determining with accuracy the operability of lung cancer. And finally, his observations opened up new opportunities for pharmacodynamic studies on the lesser circulation.

You can see, Ladies and Gentlemen, that our selectee really exemplifies the axiom that science is always in search of the truth. Very few of medical sciences have the good fortune and distinction of attaining historical stature during a lifetime. The man selected for the College medal is one of them. Of course, he is. He himself has made medical history.

Of the exceptional honors already bestowed upon him, I should like to mention that he is Officer of the French Legion of Honor, Commander of the Belgian Order of Leopold and Doctor Honoris Causa of the University of Paris. All the fine recognitions have been extended to him because of his brilliant scientific achievements, because of his tremendous contributions to the medical literature in the form of numerous articles and books, because he is a dedicated teacher of medicine but first of all, because he originated and introduced into medical practice angiopneumography.

Ladies and Gentlemen, I consider it a great privilege indeed, to present to you the recipient of the 1960 international medal of the College, the eminent Professor of Chest Diseases at the University of Lisbon, Portugal, Doctor Lopo de Carvalho.

\*Presented at the Sixth International Congress on Diseases of the Chest, Vienna, Austria, August 27-September 1, 1960.

\*\*Chairman, Council on International Affairs, American College of Chest Physicians.

## LOPO DE CARVALHO

### BIOGRAPHY

Fausto Lopo Patrício de Carvalho was born in the town of Guarda, Portugal, May 15, 1890. He was the son of Dr. Lopo de Carvalho, one of the pioneers in the fight against tuberculosis in Portugal and founder and Director of the Souza Martins Sanatorium. The first steps in his career as a specialist in chest diseases, the subject to which he has always devoted himself, were taken under his father's guidance.

He studied at the University of Coimbra and took his science degree in 1913, attaining an M.B. (19 marks out of a possible 20). In 1916, he took his degree in medicine, this time with 20 marks out of 20. He was Assistant Professor on the two faculties. After completing his medical studies, he worked for a time at the Guarda Sanatorium, where he prepared his thesis for a Doctor's degree, entitled "Artificial Pneumothorax."

In 1921, Dr. Lopo de Carvalho was one of the candidates for a post on the teaching staff in the Faculty of Medicine. He came out with top marks and was entrusted with the Chair of Medical Propaedeutics. In 1927, he moved to the Lisbon Faculty of Medicine where he continued teaching the same subject until 1934, in which year the Chair of Chest Diseases was created and he was appointed to it.

Simultaneously with his work as Professor, he was President of the National Assistance of the Tuberculous for eight years, and President of the International Union Against Tuberculosis for thirteen years. For his services in connection with these bodies, he was awarded the Portuguese Order of Public Instruction and the Legion of Honour.

Dr. Lopo de Carvalho is a member of many scientific societies throughout the world and has published innumerable books and articles.

### ANNUAL MEETING, BOARD OF REGENTS

The following resolutions, reports and elections were approved by the Board of Regents of the College at its meetings in Miami Beach, Florida, June 8 and 12:

#### Resolutions

WHEREAS, Many distinguished Fellows of the College have rendered valuable services as chairmen of councils and committees, and  
WHEREAS, Many of these officials, after a stipulated period of service, wish to relinquish the chairmanship of their respective councils and committees,  
THEREFORE BE IT RESOLVED, That in cases where chairmen of councils and committees have served the College for a period of ten years or more and discharged their duties to the credit of the organization, they be awarded a Certificate of Merit upon their retirement from office.

The following resolution was submitted upon the recommendation of Dr. Donald R. McKay, Buffalo, and Dr. Coleman B. Rabin, New York, N. Y.:  
BE IT RESOLVED, That for the purpose of establishing closer liaison between College Chapters and the national organization, the Governor of the College in each state where a chapter of the College has been organized, as well as the Regent of the College in the district, be recognized as ex-officio members of the Executive Committee of their respective chapter and that the Governor and Regent are to meet with the members of the Executive Committee of the chapter to give them the benefit of their experience and to acquaint them with the national and international programs of the College.

BE IT FURTHER RESOLVED, That this resolution be referred to the College Committee on Bylaws for appropriate action.

#### Elections

Dr. Donald R. McKay, Buffalo, New York, to the Committee on Nominations.  
Dr. Alfred Goldman, St. Louis, Missouri, re-elected a member of the Executive

#### Council.

Dr. Richard H. Overholt, Boston, Massachusetts, and Dr. William B. Bean, Iowa City, Iowa, re-elected members of the Editorial Board for *Diseases of the Chest*.

Dr. Arthur M. Olsen, Rochester, Minnesota, re-elected Chairman of the Board of Regents.

Dr. Irving Willner, Newark, New Jersey, re-elected Vice-Chairman of the Board of Regents.

The Board of Governors elected Dr. Alexander Libow, Miami Beach, Florida, to the Committee on Nominations.

Dr. Howell S. Randolph, Phoenix, Arizona, was re-elected Chairman of the Board of Governors.

**REPORT OF THE COUNCIL ON PUBLIC HEALTH**

The Council on Public Health of the College is alarmed by the excessive mortality occurring at times of influenza outbreaks. Particularly the rapidity of events and the effect on people with known cardiac and pulmonary conditions is noteworthy. Influenza vaccines contain antigen against known etiologic viruses and represent the best way to effectively blunt the force of an attack on influenza.

Therefore, the Council urges the members of the American College of Chest Physicians to practice routine annual immunization against influenza in the fall of the year, before the influenza season begins, in all of their patients with cardiopulmonary disease and disability, and in elderly patients and groups for whose care they are responsible.

Robert J. Anderson, Chairman

The Board of Regents of the College unanimously approved the above recommendation of the Council on Public Health.

**REPORT OF THE TREASURER****Statement of Income and Expenses for the Year Ended December 31, 1959****INCOME:**

Annual Dues		\$115,684.14
Fellowship Fees		27,589.25
Sales—		
Advertising	\$ 54,950.01	
Subscriptions	31,119.28	
Technical Exhibits	9,012.32	
Miscellaneous Services	5,759.91	
	<u>\$100,841.52</u>	
Discounts: Advertising and Subscriptions	11,384.42	89,457.10
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Interest: Savings & Loan Certificates		3,295.83
<b>TOTAL INCOME</b>		<b>* \$236,987.32</b>

**EXPENSES:**

Salaries	\$ 72,074.00	
Printing Journal	61,333.36	
Building Account	10,644.54	
Printing	8,854.52	
Mailing Journal	5,791.07	
Postage and Shipping	4,546.40	
Translations	325.00	
Officers and Committees	3,625.96	
Telephone and Telegraph	3,489.33	
Office Expense	4,097.17	
Travel—Executive Director	2,042.51	
Annual Meeting	9,948.47	
Interim Meeting	2,401.47	
International Meeting	5,801.10	
Public Relations	1,698.94	
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Public Health Counselor	487.71	
College History	11,140.25	
Library	332.07	
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Payroll Taxes	1,163.88	
Prize Essay Award	1,163.64	
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Contribution to World Medical Association	500.00	
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Maintenance of Office Equipment	216.50	
Depreciation—Furniture and Fixtures	1,975.41	
<b>TOTAL EXPENSES</b>		<b>219,626.70</b>
<b>NET INCOME</b>		<b>\$ 17,360.62</b>

RALPH H. MARCUS, Certified Public Accountant, Chicago, Illinois

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## COLLEGE INTERIM SESSION

The Interim Session of the College will be held at the Shoreham Hotel, Washington, D. C., November 26-28, 1960, and the American Medical Association will hold its Clinical Meeting in Washington, November 28 through December 1.

An excellent scientific program, arranged by Dr. Joseph W. Peabody, Jr. and the members of his Scientific Program Committee, will be presented on Saturday, November 26 and Sunday, November 27. In addition to formal papers and panel discussions there will be three round table luncheons each day. On Sunday evening there will be a cocktail party and banquet followed by the always popular Fireside Conferences. The semi-annual meetings of the Board of Regents and Board of Governors of the College will take place on Monday, November 28.

The complete scientific program for the Interim Session appeared in the October issue of the journal and additional copies of the program may be obtained by writing to the Executive Offices of the College in Chicago. An advance registration and order form may be found in the October journal. For hotel reservations please write directly to the Shoreham Hotel, giving arrival and departure dates, and indicating that you will attend the meeting of the American College of Chest Physicians.

## BOOK REVIEWS

**The Human Lung**, by Heinrich von Hayek, M.D., Ph.D. Revised and augmented by the author; translated from the German by Vernon E. Krah, Ph.D.; Hafner Publishing Co., Inc., New York, 1960 (XII + 372 pp, 267 + 8 fig.)

Not since William Snow Miller's classic account of 1937 has there appeared in the English language such an authoritative monograph on the microscopic structure of the human lung as that which is here briefly reviewed. Just as Miller's book summarized the intensive labors of a lifetime, so Professor von Hayek of Vienna has brought to fruition the work of twenty years. While both anatomists have dealt primarily with microscopic structure, it is characteristic of the changing times that the v. Hayek approach begins with the thorax and gross anatomy of the lungs and ends with electron microscopy. More importantly, he relates structure to function. Thus in building upon the solid foundations laid by Miller and Policard, he has discussed many problems of concern to physiologists, pathologists, pharmacologists and clinicians, in so far as these problems are related to normal anatomy.

Essentially this English edition is of the vintage of the original German Edition in 1953. To be sure, the translator has introduced a brief account of the subdivisions of the bronchopulmonary segments as proposed by Jackson and Huber (1943); the author himself has added a final chapter to cover "electron microscopic findings," including references to this and related material on the bronchial epithelium; and here and there are minor changes. But otherwise the Bibliography has not been brought up to date, nor have other significant new observations and figures been added since the completion of the German manuscript in August 1952.

The English translation, by Dr. Vernon E. Krah, Professor of Anatomy at the University of Maryland, reads smoothly, for Dr. Krah speaks with the authority of one whose research field is that of pulmonary anatomy and pathology. The English Edition is more readable than the German since it is set in larger type. Thereby it exceeds the first edition in length by some 80 pages. Reproductions of half-tone illustrations are good but not quite up to the quality of the original German cuts.

Interestingly enough, Professor v. Hayek begins the account with a discussion of tension and pressure relationships of the lung and thorax, then passes to the thorax, diaphragm, parietal pleura and general structure of the lung.

Following these are chapters on the trachea, bronchial tree, lobes and segments. In listing the branches of the bronchial tree (as in Table 3, page 91), the author—for some reason not clear to the reviewer—labels the 5th segment as "anterior" instead of "medial," and Segment 7 as "medial" instead of "medial basal." Perhaps this reflects the situation of 1952 when all the data pertaining to segments had not become available. Also, one may question the query on page 92 as to whether naming (or otherwise designating) the subsegmental bronchi "has any practical value." The answer is "Yes," for two reasons. First, these are vital to the understanding of individual variations in the pattern of the bronchial tree. For such aberrations are due largely to the shift in origin of the subsegmental branches. Second, these bronchi can be identified and used by the radiologist in analyzing bronchograms. Indeed, all of them have been portrayed very clearly by Claus Esser in his "Topographische Ausdeutung der Bronchien in Roentgenbild" (Georg Thieme Verlag, Stuttgart, 1951, 1957). See also the superb atlas just published by Bloomer, Liebow and Hales, entitled "Surgical Anatomy of the Bronchopulmonary Segments" (Charles Thomas, 1960).

After this introduction to gross structure, the author settles down to the main course of this repast, leaving the "electron microscope findings" for dessert. In logical order he describes the histological structure of bronchial wall, the terminal bronchiole and alveolar (respiratory) trees and the visceral pleura; then concentrates on the blood vessels, lymphatics and nerves of the lung. All these units and their significance are covered in great detail and with superb illustrations—not merely the normal structure but its pertinence to physiological and pathological processes.

Within the limits of the review it is not possible to discuss individual items but, in conclusion, one may recommend this book unqualifiedly to all those interested in the structural basis of the lung. Congratulations are due to author, translator and publisher for this truly excellent monograph.

EDWARD A. BOYDEN  
University of Washington

**Physiology of Cardiac Surgery**, by Frank Gollan; Charles C Thomas, 1959, Springfield, Ill.; 90 pages, \$4.50.

This small (90 pages) book is the published material presented by the author in the Beaumont Lecture of the Wayne County Medical Society and offers no pretense of being a complete review of the subject. The stated objective of the publication is to present sufficient historical, experimental, and, to a lesser extent, clinical material to serve as a framework for discussion of the physiology of hypothermia and extracorporeal circulation. Certain current favorable and unfavorable clinical responses to the various methods and usages are presented with the experimental physiologic, chemical, and pharmacologic background. The discussion includes controversies relative to the theoretic and practical applications of certain findings and the resultant implications for future trial or proofs of hypotheses.

The three chapters are: (1) Hibernation and Induced Hypothermia, (2) Extracorporeal Circulation, and (3) Extracorporeal Cooling, which includes peripheral and extracorporeal circulatory cooling.

The numerous quotations, analogies, dramatic turns of phrases, and metaphors in the text are probably due to its original preparation as a lecture. Their value for emphasis does not seem to warrant the space consumed, which might well have been devoted to the discussion of certain specific clinical problems, such as delayed post-operative death.

The above-mentioned objectives are well covered, especially the historical background and the review of certain basic physiologic phenomena as illustrated by selected experiments, many of which were performed by the author and his co-workers. The conclusions based on certain of these experiments are difficult to accept from the data presented, as in Figure 15, "Anoxic Tolerance of Beating Hearts During Hypothermia by Blood Cooling, which seems statistically inconclusive.

There is doubt that the author is entitled to project certain pharmacologic effects beyond the ranges of present clinical or experimental evidence. Issue is directly taken to his concept of the general beneficial effect of quinidine on the hypothermic heart. This drug well may prevent ventricular fibrillation but notoriously can suppress pace-making properties and induce death from a standstill. The cardiac effects of both calcium and potassium are discussed with implications that carry beyond the pharmacologic experiments quoted and seem to conflict with some recent experimental data.

There are many extremely interesting facets of the various phases of cooling and extracorporeal circulation that are covered clearly and succinctly and make the book worth reading, especially by those physicians not intimately acquainted with this field. The excursions into the future are interesting, and certain of these may be prophetic. However, others seem to have little foundation in fact for the direction in which the prophecy has been launched. It would seem wiser to have utilized this space in the text for specific clinical problems attendant on the indications and results of the several technics discussed, for example, irreversible shock.

There is an adequate bibliography covering the specific portions of the field discussed. The section on extracorporeal circulation through peripheral vessels is of particular interest and worthy of the more detailed exploration of the subject. Such procedure, as indicated by the author, is now being applied, with hope of increasing success, in the therapy of other cardiovascular clinical states not requiring cardiovascular surgery, such as shock.

JOHN J. SAMPSON, M.D., F.C.C.P.

#### ANNOUNCEMENT

A continuous Postgraduate Course in Allergy of two weeks duration is being offered by the Departments of Allergy and Applied Immunology of the Temple University Medical Center and Graduate School of Medicine of the University of Pennsylvania. Sessions will be held daily at the Temple University Medical Center from 9 a.m. to 5 p.m., February 27 to March 10, 1961. The tuition fee is \$175. Dr. Louis Tuft is director of the course with Drs. George Blumstein and Merle M. Miller as associate directors. For further information and application forms, write: Dr. George Blumstein, Temple Medical Center, Philadelphia 40, Pennsylvania.

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